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ENCYCLOPEDIA AND DICTIONARY  
OF  
MEDICINE AND SURGERY



*Printed by R & R CLARK, LIMITED, Edinburgh,*

FOR

WILLIAM GREEN & SONS

1906

**GREEN'S**  
**ENCYCLOPEDIA**  
**AND DICTIONARY**  
**OF MEDICINE**  
**AND SURGERY**

**VOL. II**  
**BREAD to EAR**



**WILLIAM GREEN & SONS**  
**EDINBURGH & LONDON**





## EDITORIAL NOTE

THIS volume carries the subject-matter of the *Encyclopedia and Dictionary of Medicine* from BRE to EAR, and the same features which characterised the first volume are present in it.

In all it contains 1758 subject-headings, and these are distributed in the following manner. There are eighty articles of more than 1000 words in length, these include noteworthy contributions to the subjects of *Bronchitis, Bronchiectasis, Burns, Bursæ, Cataract, Chest, Child, Chlorosis, Cholera, Chorea, Choroid, Climate, Colon, Colour Vision, Conjunctiva, Convulsions, Cornea, Crinæism, Curettage, Cystoscope, Deafmutism, Deformaties, Dermatitis, Diabetes, Diet, Digestion, Digitalis, Diphtheria, Drug Eruptions, Dysentery, and Ear*. New articles on such recent developments of medical diagnosis and treatment as *Cryoscopy, Cyto diagnosis, and Dechlorination* have been added, and *Dermatitis Traumatica et Venenata in Coal-Miners* is dealt with in a special contribution.

Then, there are two hundred articles of less than 1000 words, but of more than 10 lines (from 80 to 900 words) in length, these deal with a great variety of interesting subjects, such as *Biomism, Brist, Burial-Places, Calcium, Calcification, Canal Boats, Carbolic Acid, Census, Chloroma, Characteræ, Cloudy Swelling, Coccygodynia, Colles' Law, Colporrhæphy, Coma Vigil, Consanguinity, Cremation, Cyclopus, Dactylolysis, Decline of the Birth-Rate, Diazo-Reaction, Dromothelapsy, etc, etc*.

Finally, there are 1478 short articles and headings, varying in length from 10 lines to a few words. Most of these are of the nature of definitions, while others are references which serve as definitions. I have given special attention to this, the dictionaryal, part of the work, and have endeavoured to include all the terms in everyday use in Medicine at the present time, in response to a number of requests from readers of the *Encyclopedia*. I have, in some cases in which there seemed to be a necessity for it, added the derivation of the less familiar words, although of course the teaching of etymology is not the function of this series of volumes.

I may again be permitted to draw the reader's attention to the value of the cross-references, which are very numerous and, I trust, quite exact. If he will, for instance, turn up such a word as *DIABETES* (on page 319), or *CEREBRUM* (on page 80), or *COLIC* (on page 174), and simply read over the cross-references under these entries, he will find his memory refreshed on many matters relating to these subjects, and may even discover what he wanted to know without actually referring to any one of the articles quoted. Again, under the heading *DISEASE* (on page 404) will be found a list of nearly one

hundred maladies which are often distinguished by the addition of the name of the medical man who first described or who specially investigated them, with the briefest of definitions attached to them

Medicine is advancing with such rapidity in these days, and details are multiplying so quickly, that it is difficult for any work to keep pace with it and avoid omitting new terms or methods of treatment, but the fact that the present volume contains a description of the *Drummond-Morison Operation*, definitions of *Dichotomy*, of *Delta*, of *Complement*, of *Cleidotomy*, of *Dæstrum*, and of *Cordentery*, and articles on *Cytodiagnosis*, *Cryoscopy*, and *Dechlorination*, will show that I have at any rate striven to fulfil my duties as Editor in this respect also

J. W. BALLANTYNE

*September 20, 1904*

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# ENCYCLOPEDIA AND DICTIONARY OF MEDICINE AND SURGERY

**Bread.** See INVALID FEEDING (*Diet during Convalescence*), PHYSIOLOGY, FOOD AND DIGESTION (*Cereals*)

**Breakbone Fever.** See DENGUE

**Breast.** See MAMMARY GLAND, DISEASES OF. See also BANDAGES (*the breast*), CHEST, DEFORMITIES OF, INFANT FEEDING (*Human Milk, Breast-Feeding, Weaning, Wet-Nursing*), MENSTRUATION (*Vicarious*), PHYSIOLOGY, EXCRETION (*Milk-Secretion*), PREGNANCY, PHYSIOLOGY (*Changes in the Mammary*), PREGNANCY, DIAGNOSIS, PREGNANCY, MANAGEMENT, PUERPERIUM, PHYSIOLOGY (*Care of Breasts*), PUERPERIUM, PATHOLOGY (*Affections of Breasts and Nipples*), SYPHILIS

**Breastpang.** See ANGINA PECTORIS

**Breast Pump.** See PUERPERIUM, PHYSIOLOGY, PUERPERIUM, PATHOLOGY (*Affections of Breasts and Nipples*)

**Breath.** See also ALCOHOLISM, BROMISM, DIABETES MELLITUS (*Diagnosis*), HEART, MYOCARDIUM AND ENDOCARDIUM (*Stomach Symptoms, Acetone Odour*), NOSE, EXAMINATION OF (*Odour*), ESOPHAGUS (*Dilatation of*), RESPIRATION, STAMMERING, STOMACH, DISEASES OF (*Symptomatology, Foul Breath*), TOXICOLOGY (*Phosphorus, Garlic Odour*), TYPHOID FEVER (*Symptoms*)—The expired air which is popularly known as the breath consists of atmospheric air returned from the lungs saturated with watery vapour, and otherwise altered chemically and physically by having increased its proportion of carbonic acid gas, having become warmer, and being charged with more or less putrescible organic matter which in certain diseases gives it an offensive odour, as in some forms of dyspepsia, in ozæna, tonsillitis, bronchiectasis, phthisis, pulmonary gangrene, and typhus fever. It normally con-

tains 4.782 vols per cent less oxygen and 4.38 vols per cent more CO<sub>2</sub> than ordinary air. These figures give the respiratory quotient as

$$\frac{\text{CO}_2}{\text{O}_2} = \frac{4.38}{4.782} = 0.905, \text{ but this may vary under}$$

normal conditions. These chemical changes are the immediate result of the exchange of gases in the lungs, and remotely depend upon the vital exchange which takes place in the tissues (*vide "Respiration"*). Expired air also contains traces of ammonia from the blood and traces of H and CH<sub>4</sub>, probably derived from the digestive tract, whence also come such products of decomposition as acetone, which gives a peculiar and characteristic odour to the breath. This odour has been most often observed in diabetes, but as acetone is found in the urine after taking alcohol, in pneumonia, measles, and other acute diseases, especially in children, it is unsafe to attach to it any great diagnostic value, but undoubtedly its presence would suggest the desirability of a careful examination of the urine.

Sulphuretted hydrogen, if formed in the stomach or small intestine, is excreted through the lungs, as are the vegetable volatile sulphides found in onions and garlic. Foul-smelling breath is perhaps most often dependent upon putrefactive processes in carious teeth or among the papillae of a coated tongue. The normal temperature of the breath is 36.3 C., which is very near the temperature of the body. External cold increases the intake of oxygen and the discharge of carbon dioxide, while a rise of external temperature is followed by a diminution of both. Muscular exercise acts like cold, and undoubtedly a considerable part of the effect of cold is due to the involuntary movements excited by it.

Food produces the same result, while fasting lowers it. In the case of Cetti, the fasting man,

the absorption of oxygen and discharge of carbon dioxide per kilo. of body weight fell rapidly, so that the respiratory quotient which before the fast was 0.75, on the third day of fasting had fallen to 0.65, and remained during the remainder of the fast between 0.65 and 0.68. Vegetable food raises the respiratory quotient to nearly unity, while on flesh diet it is about 0.74, and on a mixed diet somewhat higher, these differences depending upon the amount of carbon present in the food.

*The relations of the breath to the etiology of disease* are not perfectly clear, but it is known (a) that animals made to rebreathe the same air die ultimately of asphyxia, and (b) there is considerable volume of evidence to show that persons living in crowded and ill-ventilated rooms, where they are compelled to breathe over again air which has been already exhausted by themselves and others, become anæmic and ill, and are especially liable to become attacked by tuberculosis, (c) the tubercle bacillus has been found in the expired breath of consumptive patients, so that doubtless it may afford a channel of infection for this and for other diseases. It is believed that measles, scarlatina, diphtheria, influenza, and the infection of a common cold may be communicated by the breath—a matter of the greatest importance in reference to prophylaxis.

A good deal of discussion has taken place as to the causes of the asphyxiating property of air vitiated by respiration, and the subject is considered in the article "Asphyxia."

*Clinical Diagnosis*—Examination of the breath may be utilised for purposes of diagnosis—

(a) For the determination of death it is a common practice to apply a feather to the mouth or the nose, or the polished surface of a mirror, in order to see whether any movement of air or deposition of watery vapour takes place.

(b) The temperature of the breath varies with the body temperature—for example, falling very low in the cold stage of cholera and rising high in fever.

(c) The odour of the breath is modified by smoking or chewing various aromatic substances, or by eating certain articles of food, such as garlic, which contain strong-smelling compounds. Alcohol gives a characteristic odour to the breath, as do many substances used as drugs—for example, bisulphite, copaiba, and the mineral poisons, especially mercury. The breath of women during menstruation may have a sweet odour like that of chloroform. In uræmia the breath often smells of ammonia, which may be demonstrated by the production of thick white fumes when a glass rod dipped in hydrochloric acid is brought near the mouth, while in diabetes its odour has been variously compared to hay, apples, sour beer, and vinegar. In pyæmia and

allied conditions (septicæmia, etc.) the odour of the breath is sweet. In dyspepsia and constipation the breath is foul and sometimes almost fecal in odour, while, when stercoraceous vomiting is present, it is undoubtedly fecal. Local morbid conditions, such as dirty or decayed teeth, ozæna, chronic follicular tonsillitis, catarrh of the mouth and tongue, ulceration or gangrene, malignant disease, caries of bone in the mouth or nose, ulceration of the larynx or decomposition of retained bronchial secretion in dilated bronchial cavities, or gangrene of the lung, may give rise to the most foul odours of decomposition in the breath.

(d) Microscopical examination of the expired air has in phthisis shown the presence of the tubercle bacillus (A. Ransome), and if applied in other diseases might succeed in demonstrating other pathogenic bacteria, but this method has not so far been very extensively used. Many authorities consider that the tubercle bacillus is never found in the breath in ordinary respiration, but only when associated with the act of coughing.

(e) The same may be said for bacteriological examination, this mode of searching for microbes not having been systematically applied to the breath.

(f) Chemical examination is the only scientific method which has hitherto yielded much result. The respiratory quotient is found to vary according to the state of metabolism of the body. In inanition it is diminished, while in certain wasting diseases it is increased. There is said to be an increase of CO<sub>2</sub> in asthma and bronchitis, while in cholera there is a decrease.

In Bright's disease and uræmia the ammonia is greatly increased. Marsh-gas may be expired in such quantity as to make the breath inflammable, and several instances have been recorded where the patient has accidentally set fire to his breath, for instance, when trying to light his pipe. Such patients have generally suffered from dilated stomachs, in which decomposition of food was associated with the formation of this gas.

In diabetes the breath sometimes contains acetone. In poisoning from hydrocyanic acid this acid is present in the expired air.

*Treatment*—As evil-smelling breath is generally a secondary symptom, its treatment depends for the most part upon the removal or cure of the primary condition, and it would be out of place here to do more than indicate the lines of treatment. Where the seat of the disease is in the mouth it is accessible, and carious teeth, etc., should be stopped or removed, a coated tongue scraped or disinfected, the pharynx sprayed with an antiseptic solution, etc. For ozæna the use twice a day of a nasal douche is often the only remedy.

In acute diseases, where the mouth is foul with sordes, it should be cleaned with glycerine and rose water. The fetid odour from bronchoectatic

cavities and gangrene of the lung may be kept down by creasote taken in capsules. Dr. G. Vivian Poore has recommended garlic for this purpose, in the belief that this vegetable contains an antiseptic principle to which it owes its reputation as an article of diet. In two cases of the kind wherein a trial was made of Dr. Poore's suggestion, the stench was diminished, and was curiously enough not replaced by any offensive smell of garlic.

Putrefactive changes in a dilated stomach can be most efficiently treated with lavage by means of a soft stomach tube and a tepid solution of salicylate of soda (1-1000).

The value of hot intestinal irrigation in uræmia and similar diseases has yet to be determined, but is at least worthy of a fair trial.

**Breathing.** See CHILDREN, CLINICAL EXAMINATION OF (*Brathing*), Hysteria, PHYSIOLOGY (*Respiration*), RESPIRATION.

**Breathlessness.** See DYSPNOEA, HEART, MYOCARDIUM AND ENDOCARDIUM (*Symptomatology, Dyspnoea*), RESPIRATION, etc.

**Breech Presentation.** See LABOUR, DIAGNOSIS AND MECHANISM (*Podalic Lies*), LABOUR, MANAGEMENT OF.

**Bregma.**—The anterior fontanelle of the head in infants and the place corresponding to it in adults. See LABOUR, PHYSIOLOGY OF (*Passengers, Fetal Head*).

**Bremer's Blood Test.**—Failure of the red cells (in diabetic blood) to take the red stain as the normal corpuscles do. See DIABETES MELLITUS (*Coma, Diagnosis of*).

**Brenzkatechinuria.**—Brenzkatechin (or alkapton) in the urine. See ALKAPTONURIA.

**Brepho.**—Brepho, as a prefix, signifies "pertaining to an infant or foetus," and is used in such words as BREPHOTROPHIUM, a foundling hospital. BREPHOCACOLPIA, vulvar gangrene in infants, BREPHOPOLYCARCIA, obesity in infants, etc.

**Breweries.** See ALCOHOL (*Ber*), TOXICOLOGY (*Arsenic*).

**Bricklayer's Cramp.**—Spasm of the right hand from holding the trowel. See SPASM (*Habit*).

**Brickworker's Anæmia.** See PARASITES (*Uncinaria*).

**Brides-les-Bains.** See BALNEOLOGY (*France*).

**Bridge of Allan.** See BALNEOLOGY (*Great Britain*), MINERAL WATERS (*Muriated Saline*).

**Bridge Work.** See TEETH (*Bridge and Bar Work*).

**Brighton.** See THERAPEUTICS, HEALTH RESORTS (*English*).

**Bright's Disease.** See NEPHRITIS. See also ANÆMIA, BRAIN, BRONCHITIS, ACUTE (*Causes*), COLON, DISEASES OF (*Colitis*), CONJUNCTIVA, DISEASES OF, COUGH, EAR, OTITIS MEDIA (*Causes*), ECLAMPSIA, HEART, MYOCARDIUM AND ENDOCARDIUM (*Etiology, Muscle Failure*), HYDROPATHY, INTESTINES, DISEASES OF (*Enteritis*), LEUCOCYTOSIS, LUNGS, VASCULAR DISORDERS (*Etiology*), MENSTRUATION AND ITS DISORDERS (*Amenorrhœa*), OCULAR MUSCLES, AFFECTIONS OF, PERICARDIUM, DISEASES OF (*Pericarditis*), PERITONEUM (*Acute Peritonitis*), PLEURA, DISEASES OF (*Acute Pleurisy, Hydrothorax*), PULSE, RETINA AND OPTIC NERVE, STOMACH AND DUODENUM, DISEASES OF; TINNITUS AURIUM.

**Brim of Pelvis.** See GENERATION, FEMALE ORGANS OF (*Ovarious Pelvis*).

**Briquet's Syndrome.**—A group of symptoms, including shortness of breath, suppression of the voice, and paralysis of the diaphragm, occurring in hysteria.

**Brittleness of the Nails.** See NAILS, AFFECTIONS OF.

**Broad Ligament, Diseases of the.**

DISPLACEMENTS OF	3
INFLAMMATION	4
MESOSALPINX	
MESOMETRIUM	
TUMOURS	5
MESOSALPINX	
MESOMETRIUM	

See also ECTOPIC GESTATION, FALLOPIAN TUBES, OVARIES, DISEASES OF, PELVIS, DISEASES OF THE CELLULAR TISSUE, PUERPERIUM, PHYSIOLOGY, UTERUS, DISPLACEMENTS, etc.

THE serous fold known as the broad ligament is divided into the mesosalpinx or mesentery of the Fallopian tube and the mesometrium proper, which lies below the level of the attachment of the ovary. In disease the mesosalpinx is mainly associated with tumours, the mesometrium with inflammatory deposits.

*Displacement of the Folds of the Broad Ligament by Tumours.*—PAROVARIAN cysts, cystic tumours developed in the hilum of the ovary, and, in rare instances, tumours of the ovary proper, make for themselves a capsule of the mesosalpinx, which they greatly distend. This condition is easily recognised during operation by the position of the Fallopian tube, which is much elongated and stretched over the top of the capsule. These same tumours sometimes

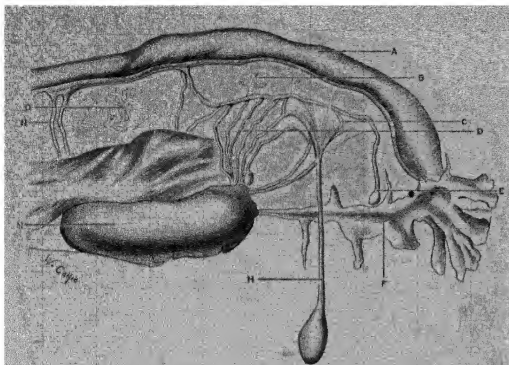


FIG. 1.

Anterior view of Left Mesosalpinx.

- A, Fallopian tube.
- B, Reliefs of mesonephros.
- C, Curved tubules of Kobelt entering into formation of pedunculated cyst, H.
- D, Vertical parovarian tubules.
- E, Sessile terminal cyst of duct of Gartner.
- F, Pen-shaped ovarian ligament.
- G, Left ovary.
- N, Tubular reliefs attached to duct of Gartner.
- O, Unattached tubular reliefs.

(Nearly natural size.)

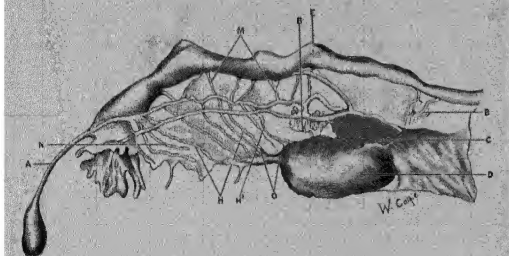


FIG. 2.

Anterior view of Right Mesosalpinx.

- A, Pedunculated terminal cyst of duct of Gartner.
- B, Reliefs of mesonephros.
- C, Cut surface of broad ligament.
- D, Ovary.
- E, Fallopian tube.
- H, Parovarian tubules.
- H', Atrophied linear remains of innermost vertical tubules.
- M, Lymphatics.
- N, Curved tubules.
- O, Rete ovarii.

(Reduced one-third.)

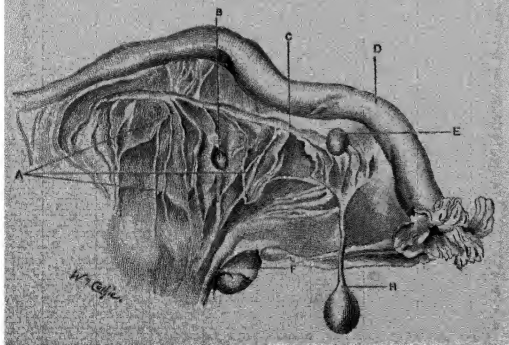


FIG. 3.

Anterior view of Left Mesosalpinx.

- A, Vertical tubules, some branched.
- B, Sessile cyst connected with vertical tubule.
- C, Duct of Gartner.
- D, Fallopian tube.
- E, Sessile terminal cyst of duct of Gartner.
- F, Outer extremity of ovary.
- H, Pedunculated cyst from tubules of Kobelt.

(Natural size.)

## DISSECTIONS OF THE BROAD LIGAMENT AND OF ITS CONTENTS

(After BALLANTYNE and J. D. WILLIAMS)



toma may burst Fatal intraperitoneal hemorrhage is in either case very probable Should the hæmatoma remain stationary, it is not likely to suppurate The hæmatoma associated with *Extra-uterine Pregnancy* is described under that heading (*vide "Ectopic Gestation"*)

**TUMOURS OF THE BROAD LIGAMENT** (1) *Of the Mesosalpinx*—When the uterus is the seat of a large fibro-myoma, and in certain other conditions involving obstruction to the lymphatics of the pelvic viscera, large yellow blebs or bullæ of irregular form are often seen covering the mesosalpinx These "lacunar cysts" or "sub-serous hygromas" do not in themselves endanger the patient, nor do they interfere much with operative manipulations, they are simply important as usually indicating serious complications elsewhere

Anatomically the simple parovarian cyst is a true tumour of the mesosalpinx, and so are all papillomatous cysts developed from the parovarium, and from that portion of Gartner's duct which lies within its folds The different ways in which the folds of the mesosalpinx may be opened up by tumours from other parts is described above

*Small fatty tumours*, pedunculated or sessile, have been detected in the mesosalpinx, which often contains a little fat Paron's case of lipoma of the tube was possibly a fatty tumour of the mesosalpinx surrounding the tube

(2) *Of the Mesometrium*—This part of the broad ligament may be the seat of a *fibroma* or *fibro-myoma* developed quite independently of the uterus, from the fibrous and muscular tissue in its folds A tumour of this class may attain a great size, and press upon the vessels and nerves of the pelvis and on the ureter No other kind of abdominal tumour displaces peritoneum more widely or more freely In comparison with uterine "fibroids," these mesometric fibro-myomas develop in younger subjects, large growths of this class having been removed from women under thirty years of age

"Fibroids" originating in the uterus, especially near or in the cervix, tend to burrow into the mesometrium They then lie very close to the ureter. In exceptional cases that duct has been found passing over and not under the burrowing tumour

There is often much fat in the mesometrium, and hence it is not surprising that *lipoma* of that fold has been recorded. *Sarcoma* has also been observed Freund describes a case of extensive invasion of the broad ligament with *echinococci*

**Broadbent's Law.**—The hypothesis that bilaterally associated movements are represented on both sides of the brain, and that the closer the bilateral association the more nearly equal is the representation on the two sides of the brain See BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Paralysis from Vascular Lesions*)

**Broadbent's Sign.**—One of the diaphragm-phenomena in adherent pericardium, a visible retraction of the thoracic wall, well seen on the left side posteriorly between the eleventh and twelfth ribs, it is due to the systolic tug of the heart communicated to the chest wall along the lines of attachment of the diaphragm

**Broca's Convolution.** See APHASIA (*Anatomical Introduction*), BRAIN, PHYSIOLOGY OF (*Functions of Cerebral Center*), PHYSIOLOGY, NERVOUS SYSTEM (*Cerebrum, Discharging Mechanism*)

**Brodie's Abscess.** See KNEE-JOINT, DISEASES OF (*Pyogenic Diseases, Chronic Osteomyelitis*)

**Broiling.** See BRANDERING, INVALID FEEDING (*General Preparation of Meats*)

**Brom-**—As a prefix Brom- indicates the presence of *bromine* in various chemical compounds, such as Bromacetal, Bromacetamide, Bromacetic Acid (mono-, di-, and tri-), Bromacetones, Bromacetyl, Bromacetylene, Bromacrylic Acid, Bromadipic Acid, Bromalbumin, Bromaldehyde, Bromalazine, Bromallyl, Bromaloin, Bromamide, Bromanylene, Bromamine, Bromanisol, Brombenzene, Brombenzoyl, Bromcaffein, Bromcannamine Acid, Bromethyl (Hydrobromic Ether), Brometone, Bromhydium, Bromhydroquinone, etc

**Broma.**—From the Greek *βρῶμα*, signifies food, especially solid, also a proprietary preparation (like chocolate), also as a prefix in such words as BROMATOLOGY (dietetics), BROWOGRAPHY, and BROMATOMETRY

**Bromal Hydrate.**—A substance, crystalline in character, similar to chloral hydrate, acting as a narcotic, and tending to slow the pulse and the respiration, used in epilepsy, chorea, etc, in doses of 1 to 7 grains

**Bromalbumin.**—A preparation used in epilepsy, etc, and consisting of bromine and albumin

**Bromatology.** See DIET

**Bromatoxism.**—Poisoning by food, of allantois, botulism

**Bromhidrosis.** See BROMIDROSIS

**Bromide.** See BROMUM.

**Bromidia.**—A proprietary preparation, containing chloral hydrate, bromide of potassium, and the extracts of cannabis indica and hyoscyamus, it acts as a hypnotic

**Bromidin.**—A proprietary preparation, acting as a hypnotic, and said to consist of chloral, cannabis indica, and hyoscyamus.

**Bromidrosis.**—A diseased condition of the skin, in which the sweat assumes a fetid odour (*βρῶμος*, a stench). In *bromohyperidrosis* the sweat is excessive in amount as well as offensive in odour. *See SKIN, DISEASES OF SWEAT AND SEBACEOUS GLANDS*

**Bromine.** *See BROMUM*

**Bromiodoform.**—A substitution compound of bromum and iodoform

**Bromipin.**—A solution of bromum in oil of sesame, used as a hypnotic in epilepsy, chorea, neurasthenia, and hysteria (dose, half to one teaspoonful)

**Bromism.** *See also EPILEPSY (Medicinal Treatment) PHARMACOLOGY. TOXICOLOGY*

The term bromism is applied to the series of symptoms that are met with resulting from the excessive use of one or more of the bromine compounds. Since the discovery of the sedative influence of the bromides on the central nervous system, these remedies have been very extensively used for many nervous disorders, and it is especially in chronic nervous cases, *e.g.* epilepsy, that the untoward effects of the bromides are seen. The degree of tolerance for bromides varies, as for other medicinal remedies. In some cases the continued use of even small doses—5 to 10 grams—leads to a manifestation of the minor or major symptoms of bromism, while in other cases very large doses are tolerated even for lengthened periods. (Clouston records an interesting case (vol. 1 p. 58) of a boy of eleven tolerating 60 grams daily for two years. The nature of the disease and the idiosyncrasy of the patient are the important modifying factors)

The depressing influence of the bromides on nerve and muscle cells, taken with their irritant property during excretion by the usual channels, is responsible for the symptoms of bromism. These may be briefly summarised as follows:—

**Neuro-muscular System.**—A diminished capability for physical or mental work, with occasional headache, which may be of a severe character and chiefly in the frontal region, are the earlier symptoms. Later, slight forgetfulness, diminished mental capacity, general weakness of mental faculties, diminution of the palatal and pharyngeal reflex, and in still later stages some disturbance of gait may be present. In not a few cases, however, especially in epileptics, the first symptoms are those of an increased excitability of the brain cortex, amounting even to mania (Bromomania). The development of any such symptom in these cases should therefore lead the practitioner to a careful survey as to the doses and duration of the treatment

**Cardio-vascular System.**—Along with the general muscular depression there is more or less

evidence of general cardiac enfeeblement. The heart's action is weak and the pulse feeble. The rate may be slightly diminished or increased. A few cases are recorded where death has been attributed to the stoppage of the heart in diastole as the result of excessive use of the drug. Other indications of vasomotor disturbance may be present, *e.g.* cold extremities and clammy state of the skin

**Cutaneous System.**—An acne eruption, most marked on the forehead, neck, face, and trunk, is frequently the earliest symptom to attract attention. In other cases the eruption may be erythematous, scarlatiform, pemphigoid, or even rupial in nature. In children the eruption may pass from a discrete acne type to take on a papillomatous appearance

**Other Systems.** The more important symptoms encountered in the other systems are loss of appetite, foul breath, anemia, dilatation of the pupil, diminution of the respiratory rate, and slightly depressed temperature

**Treatment.**—Prophylaxis is all important. A full recognition of the pharmacological actions and careful observation of patients using these remedies, noting especially the condition of the skin, mucous membrane, and general vital capacity, will suffice in all cases to prevent the development of the condition in any great degree

Cumulative treatment resolves itself into a stoppage of the use of the drug, strict attention to the action of the skin, kidneys, and bowels, and the use of general tonic remedies. Hydrotherapeutic measures are of great assistance, both from their local and general effects. Small doses of arsenic given in combination with the bromides are of some service in diminishing the tendency to bromism. Iodine arsenicals is the form most commonly used, but some writers believe that arsenate of soda is the most useful preparation, being less likely to produce any gastric intestinal irritation

Cannabis indica is also frequently used along with the bromides with the object of diminishing the tendency to the production of untoward disturbances of the nervous system

**Bromocoll.**—A combination of bromine, tannin, and gelatine, it is thought that the bromine is not set free till the medicine reaches the intestines, it has been recommended in epilepsy and (externally) in lichen and eczema

**Bromoform.**—An anæsthetic and hypnotic medicine ( $\text{CHBr}_3$ ) having reactions similar to chloroform

**Bromol.**—A proprietary preparation, disinfectant and caustic, tribromophenol

**Bromomania.** *See BROMISM*

**Bromomenorrhœa.**—An offensive condition of the menstrual discharge. *See MENSTRUATION AND ITS DISORDERS*

**Bromopyrin.**—A mixture of antipyrine, caffeine, and bromide of sodium

**Bromorcin.**—A bromine substitution compound of cinch, which is a homologue of resorcin

**Bromum.** See also ANÆSTHESIA (*Bromide of Ethyl*), BROMISM, CONVULSIONS, INFLAMMATION, DRUG Eruptions (*Bromine*), EPILEPSY, HYPNOTICS, PHARMACOLOGY, etc.—The action of *bromine* is similar to that of chlorine and iodine. It is rarely used in medicine, and is of importance solely as the source of the bromides. 1 *Potassu Bromidum* is composed of colourless glassy cubes, freely soluble in water. Dose—5-30 grs. 2 *Sodu Bromidum* is a powder made up of small white cubic crystals, soluble 1 in 2 of water. Dose—5-30 grs. 3 *Ammoni Bromidum* consists of small colourless cubic crystals, soluble 1 in 1½ of water. Dose—5-30 grs. 4 *Acidum Hydrobromicum Dilutum*, a colourless liquid prepared from potassium bromide. Dose—15-60 m. Bromides are administered whenever it is wished to diminish reflex excitability of the nervous system. Formerly they were applied in strong solution directly in pharyngeal diseases associated with spasm, and before making a laryngeal examination, but cocaine has taken their place for these purposes. In nervous diseases, accompanied by convulsions, their action is almost specific. For epilepsy, gradually increasing doses of potassium bromide should be given until a dose is reached which gives the maximum benefit, and then the administration may be continued indefinitely. A good way is to give the whole daily amount in one dose at bedtime. Sometimes as much as 2 drs. per day may be required, but no fixed rule as to dosage can be laid down. When large doses have to be given it is well to order a mixture of the three bromides. In most cases a diminution of the number of seizures is all that is achieved, but should the fits disappear altogether the use of the bromide must be continued for months in the hope that a cure may be effected. Bromides have little effect on *petit mal*. In tetanus very large doses are required if any good is to result—say 60 grs. every two hours. In delirium tremens bromide is invaluable, and is usually administered in conjunction with chloral hydrate. In cases of sleeplessness due to worry, overwork, or climacteric disturbances a small dose of a bromide is a most efficient hypnotic, but it is of little service if the insomnia be caused by pain or organic disease. "Bromidia," a proprietary mixture consisting of potassium bromide, chloral, camphis indica, and hyoscyamus, is much used as a hypnotic. In the severe headaches of migraine, bromides may be the only drugs capable of affording relief and they are also useful in some forms of neuralgia. In children they are much used for convulsions of a

reflex nature, but they are contra-indicated in chorea, unless the movements are excessively violent, or are associated with sleeplessness or nocturnal restlessness. Ammonium bromide is useful in whooping-cough. Bromides are recommended for palpitation, especially when due to functional disorders of the heart. They are indicated in cases of incontinence of urine, the result of spasmodic contraction of the bladder. They are also very efficient anaphrodisiacs. *Acidum Hydrobromicum Dilutum* is similar in action to the bromides, but cannot be taken over such a long period, on account of the disturbance of digestion caused by the acid. It has been strongly advocated for trifling heart disorders associated with palpitation and uneasiness, and is said to relieve noises in the ears in some cases. Bromides of *calcium*, *lithium*, and *strontium* have been used at various times, but they have no real advantages over the official salts.

**Bronchadenitis.** Inflammation of the bronchial glands. See BRONCHI, BRONCHIAL GLANDS.

**Bronchi, Diseases of.**—The affections of the bronchi and associated glands are described in the following articles:

1. **Bronchial Glands**
2. **Bronchitis** (for Capillary Bronchitis see "Pneumonia")
3. **Bronchiectasis**
4. **New Growths.** See "Lungs" and "Mediastinum."

#### Bronchial Glands

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See also COUGH (*Clinical Varieties*), LUNG, TUBERCULOSIS (*Pathological Anatomy*), LUNGS, ABSCESS OF, LYMPHATIC SYSTEM, PHYSIOLOGY AND PATHOLOGY, LIVER (*Abscess of*), MEDIASTINUM.

**ANATOMY.** The glands may be classified into three main groups (Baréty)—

- (1) Those around the lower end of the trachea and the main bronchi—the peri-tracheo-bronchial group.
  - (2) Those between the main bronchi, chiefly beneath them—the inter-tracheo-bronchial group.
  - (3) Those between the divisions of the bronchi—the inter-bronchial group.
- These three groups are in connection with—
- (1) The superficial and deep lymphatics of the lungs,
  - (2) The lymphatics of the visceral pleura
  - (3) Some of the lymphatics of the œsophagus.
- These lymphatics communicate freely



The glands themselves vary a good deal in size and shape, and the inter-bronchial group extend for some distance into the substance of the lung, according to Crivellier, as far as the fourth divisions of the bronchi. The right bronchus is shorter than the left, and is more directly a continuation of the trachea than is the left, and there are a larger number of glands in connection with the right than with the left bronchus.

From the position of the glands it will be seen that they come into relation with the following intrathoracic structures—

The trachea, the bronchi as far as their fourth division, the lungs, the pulmonary arteries and veins, the superior vena cava, the innominate, subclavian, and axillary veins, and the aorta, the vagus nerve and its recurrent branches, and the pulmonary plexuses, the pericardium, and the œsophagus.

The tracheal bifurcation corresponds in level to a horizontal line drawn through the junction of the manubrium and body of the sternum, this corresponds behind to the spine of the fourth dorsal vertebra. The horizontal level at the point of intersection of the lines corresponding to the spines of the scapulae will also give us the level of the tracheal bifurcation.

**MORBID ANATOMY AND PATHOLOGY.**—The bronchial glands may be the seat of various morbid changes, either as a primary condition, or secondary to changes in the neighbouring parts from which the lymphatics pass to the glands, or as part of some general condition affecting the lymphatic glands.

The most common morbid condition is *pigmentation*, and in adults this condition is practically constant. Next to pigmentation, *hyperæmia* and *inflammatory swelling* of the glands are the most common morbid conditions, and are met with in association with similar conditions in the pleura, lungs, and bronchi. The amount of swelling may be considerable, the glands being three or four times their normal size, pinkish grey in colour and succulent, or, in cases where the swelling is marked, becoming pale grey and medullary in appearance. The enlargement is not so important as the accompanying lowered activity of the glands, which results in diminished resistance to invasion of the tissues by micro-organisms, more especially by the bacillus tuberculosis. It is quite probable that owing to the retardation of the lymph stream in the lungs or pleura, the bacilli have a longer opportunity of getting into situations suitable for their growth and multiplication.

In this way the frequency of tuberculous disease of the glands after measles and whooping-cough may be explained. That these changes in the glands are not necessarily secondary to tuberculous disease of the lungs or pleura is evidenced by the fact that we often find the changes in the lungs recent while those in the

glands are obviously of long standing, and if we find in a given case the region of the root of the lung to be the part most affected, it is most probable that the disease originated in the glands and spread outwards. The frequency of caseation of the bronchial glands is shown by the fact that in a series of 300 post-mortems made on children under twelve years of age the writer found the bronchial glands caseous in 110, i.e. in 37 per cent of the cases. Such glands may undergo various changes, becoming fibrosed or calcified, or they may undergo softening or suppuration.

Caseation of the glands is usually associated with a tuberculous lesion in the lungs or elsewhere, but may be the sole tuberculous manifestation present. An analogous condition is met with in the abdomen when we find caseous mesenteric glands as the sole tuberculous lesion. The writer believes that caseous bronchial glands are more common than caseous mesenteric glands, thus indicating that tuberculous infection takes place more frequently through the air-passages than the alimentary tract.

Apart from tubercle, metastatic deposits in the glands are of frequent occurrence in association with malignant deposits in the lungs, pleura, and œsophagus either of a primary or secondary nature.

Primary new growths in the glands are most commonly of the nature of sarcomata, especially lympho-sarcomata (see "Mediastinum").

In lymphadenoma (*q.v.*) the bronchial glands take part in the general glandular enlargement.

In both secondary and tertiary syphilis the bronchial glands may share in the general glandular enlargement.

When waxy disease is widespread the glands throughout the body may be the seat of deposits of waxy material, but it is very doubtful if any distinct symptoms or physical signs can be traced to this condition in the bronchial glands.

Disease of the bronchial glands produces effects in two ways—

- (i) By impairment of function
- (ii) By the production of enlargement with consequent pressure effects.

The effects of impaired function have been briefly referred to above, and are more fully dealt with under the head of tuberculous. Practically, enlargements of the bronchial glands sufficient to cause pressure effects are due to either tubercle or new growths, and the former is infinitely the more common in children, the latter in adults. Although disease of the bronchial glands is not uncommon in adults, it is chiefly in children that definite symptoms are caused, and the subsequent remarks refer chiefly to them.

Pressure effects may be exercised on the following parts—(i) The trachea and bronchi, (ii) the lung, (iii) the blood-vessels, (iv) the nerves, (v) the œsophagus, (vi) the pericardium.

(1) *Pressure on the Trachea and Bronchi*

Some degree of narrowing or displacement is not infrequently seen. In other cases inflammatory adhesions with the wall of the trachea or bronchus develop, and in this way a path is opened up for the infection of the air-passages with tubercle. Perforation of the air-passages is not uncommon. In 800 autopsies on children it was present in 25 cases, i.e. over 3 per cent. The right bronchus is more frequently perforated than the left. This may be due to the shorter length of the right bronchus, and to the larger number of glands on that side. The trachea is less frequently perforated than the bronchi. After perforation the softened contents of the glands may be discharged, and the perforation closed by cicatrization, the scar left being small and not tending to cause stenosis of the tube as in cicatrization of syphilitic lesions. When perforation occurs there is a liability to the occurrence of hæmorrhage from erosion of vessels, but this is rarely so severe as in the cases where the lung itself is involved. Fatal asphyxia may follow the impaction in the glottis of a large caseous mass in process of discharge. Mediastinal abscess with singular emphysema followed perforation in one case of the writer's, reported in the *Practitioner*, June 1895.

(n) Pressure on the lung itself results in inflammatory adhesions, and in this way the lung tissue becomes involved, and infiltrated with caseous material which tends to soften and form cavities. These are not uncommon in the lower lobes of the lung. Malignant disease of the bronchial glands usually extends inwards along the bronchi, compressing them, and frequently producing a bronchiectatic condition of the lung.

(m) The effects of pressure on the blood-vessels show themselves chiefly on the veins of the head and neck, which become over-filled, and these parts become congested and even œdematous. The smaller veins may have their walls invaded by tubercle, and thus become the means of producing a generalised tuberculosis. Erosion of branches of the pulmonary artery and vein in the lung may give rise to profuse and even fatal hæmorrhage. The arteries are much less commonly affected, though cases are on record where a crataecous gland has ulcerated into the aorta and produced fatal hæmorrhage.

(iv) The effects of pressure on the nerves in the case of caseous bronchial glands are very difficult to distinguish from those due to pressure on the air-passages themselves, but there are, at the same time, cases where pressure on the recurrent laryngeal nerve of one side has produced unilateral paralysis of the vocal cords. The vomiting which occurs in association with enlarged glands, as well as in some cases of pulmonary tuberculosis, has been attributed by some to the effects of pressure on the branches of the vagus nerve. Malignant growths in the

bronchial glands may, however, produce marked pressure effects on the nerves (*vide* "Mediastinum").

(v) The effects of pressure on the œsophagus are evidenced by some narrowing of that tube and consequent dysphagia. Adhesions may form between the glands and the œsophagus, and if cicatrization occurs the wall of the œsophagus may be dragged on, and thus a traction diverticulum result. Perforation of the œsophagus by a caseous gland is not of very rare occurrence in children, in examining the records of over 3000 cases in children there were thirteen such cases or about 0.4 per cent. Perforation of the œsophagus may be accompanied by profuse and even fatal hæmorrhage. When rupture into the air-passages occurs at the same time, gangrenous processes are liable to be set up in the lungs or mediastinum.

(vi) Pressure on the *pericardium* may cause adhesion between the pericardial sac and the glands, and perforation into the pericardial sac may follow, and then a tuberculous or suppurative pericarditis may be set up. Perforation of the pericardium takes place, most commonly in the upper part of the serous sac.

In addition to these events we may find a combination of two or more of the above-mentioned conditions occurring simultaneously. The writer has seen a case where caseous bronchial glands had perforated the œsophagus, right bronchus, right lung, and the pericardium.

*SYMPTOMS AND PHYSICAL SIGNS*—Enlarged bronchial glands may exist without giving rise to any symptoms, and to physical signs of very little diagnostic value.

The chief symptoms produced by them are cough, vomiting, dyspnoea, dysphagia, and pain.

*Cough*—This is paroxysmal in character, and closely resembles that of whooping-cough. As a rule it is unaccompanied by any expectoration or whoop, but when severe may be accompanied by vomiting.

When the trachea or bronchi are being invaded by caseous glands there is usually some tracheitis and bronchitis, and this is not infrequently accompanied by hæmoptysis varying in amount from a mere streak of blood to profuse or even fatal hæmorrhage.

In some cases the cough has a distinctly clanging character. The voice, as a rule, is not altered, but sometimes the cry has a distinct stidor. The paroxysms of coughing are sometimes so severe that after them the child will fall back quite exhausted. Epistaxis may accompany a fit of coughing, but much less commonly than cyanosis. When a caseous or crataecous mass ulcerates through the air-passages, in addition to the hæmoptysis mentioned, the caseous or crataecous mass may be expectorated.

*Vomiting* is present in some cases, and generally accompanies severe attacks of coughing.

When the œsophagus is involved there may be hæmatemesis, and the passage of blood in the stools, or if the air-passages are involved at the same time, food may be expectorated.

The breath in a large number of cases of ulceration into the air-passages has a very offensive smell, though factor of the breath does not necessarily indicate ulceration into the œsophagus.

*Dyspnea* is sometimes well marked, the patient being unable to lie down in bed, and dreading any movement or interference such as for feeding or changing.

The dyspnea in some cases is more of the nature of an expiratory than an inspiratory dyspnea.

When the recurrent laryngeal nerve is involved by a new growth in the bronchial glands the vocal cords may be paralysed (*vide* "Mediastinum").

*Dysphagia* may be associated either with pressure of enlarged glands on the œsophagus, or with actual ulceration of a caseous gland through the œsophageal wall. It is not a very common symptom.

*Pain* when present is generally referred to the region of the manubrium, or less commonly to the epigastrium. When the œsophagus is involved there may be some pain in swallowing, although extensive ulceration of that tube may be present without giving rise to any symptoms.

The physical signs produced by enlarged bronchial glands are often masked by the signs of other concurrent disease in the chest.

*Inspection.* The face is frequently cyanosed, especially after an attack of coughing, and there is often marked dyspnea with recession of the lower part of the thorax; this recession is, as a rule, bilateral in small children, but in older children in whom the thoracic parietes are firmer there may be distinct unilateral recession. The lower part of the sternum is often retracted during inspiration. The veins over the front of the chest are frequently enlarged. A moderate amount of œdema of the face may be present.

*Palpation.* In addition to confirming the results of inspection, may reveal the presence of enlarged glands in the neck or axillæ. The presence of surgical emphysema may also be verified. Vocal fremitus, or in small children the cry fremitus, may be diminished over the area supplied by a compressed bronchus, or increased over the manubrium.

*Percussion.* Impairment of resonance of the percussion note may be present over the manubrium and inner end of the first or second intercostal spaces anteriorly, and similarly in the suprascapular fossæ and interscapular region posteriorly. The percussion of the back is much less reliable in small children than in adults. Special care should be taken in examining small children to see that the

child sits up or is placed quite straight, and not allowed to lie over to one side, as it is so apt to do when being held by the mother or nurse.

*Auscultation.*—The breath sounds over the lungs may be simply weak, while over the dull area there is often marked tubular or bronchial breathing, and in some cases there may be almost amphoric breathing. Vocal resonance over the dull area is increased, and may have an ægophonic character. When catarrh of the air-passages exists there are the usual signs of this condition, and should these be unilateral in distribution, suspicion should be directed to the bronchial glands. The usual sequence of auscultatory signs in the lungs is weak breathing followed by bronchial breathing, to which fine crackling râles, and eventually sonorous and bubbling sounds, are added.

Where the lung is involved either in tuberculous processes or by the formation of breaking-down areas, these signs become masked by those of pulmonary tuberculosis, or emphysema, or the signs of bronchiectasis.

Eustace Smith has pointed out that a pitting sound can be produced in the vessels of the neck during respiration by making the child look upwards, and so extend the neck and compress the vessels by the retro-mandibular glands. The stethoscope must be applied below the supra-sternal notch.

If signs of tuberculous consolidation are present and are limited to the middle lobe, this would suggest the bronchial glands as the focus (*et origo mali*).

*Diagnosis.*—It must be borne in mind that the condition frequently exists without giving rise to any symptoms, and further, that the physical signs are often equivocal. Every condition liable to produce paroxysmal cough and dyspnea must be differentiated. Of these the more important are whooping cough, enlarged tonsils, pulmonary tuberculosis, emphysema, and bronchiectasis.

In other cases asthma, a foreign body in the air-passages, adherent pleura, pressure on the air-passages by aneurysm, abscess, a new growth, prevertebral abscess in the upper dorsal region, laryngeal diphtheria, and laryngismus stridulus must be excluded. Careful consideration of the history and detailed examination of the pharynx, larynx, and chest will usually suffice to differentiate these conditions.

*Prognosis.*—When the enlargement is due to malignant disease this is necessarily hopeless, but when due to tuberculous disease it is not of necessity so grave, and yet it must be borne in mind that tuberculous meningitis not infrequently is met with in cases in which the bronchial glands are the only recognizable sites of previous tuberculous infection. Recovery is possible even when the air-passages or the œsophagus are perforated.

**TREATMENT**—The treatment of enlarged bronchial glands is that of the diseases which cause the enlargement.

Prophylactic treatment is of great importance. After an attack of measles, whooping-cough, or influenza, when all risk of infection is past, a stay at the seaside, in a part having a sheltered and southern aspect, is advisable, or else in some high and dry locality sheltered from the east wind. Clay soils are to be avoided. The clothing should be warm, woollen, underclothing being best, and the limbs should be entirely covered up. It is astonishing how much thoughtlessness is displayed in the exposure of the lower limbs of children to the air out of doors, both in health and after illness, in the erroneous belief that it is "strengthening" to them.

Special precautions should be taken with the food, all milk being boiled, unless the absence of tuberculous disease in the cows has been ascertained by inoculation.

The dwelling and sleeping rooms should be large, light, well ventilated, free from dust, and the windows should be kept open at night.

The medicinal treatment of these cases consists in the administration of some form of cod liver oil in combination with iron, or maltine, or the hypophosphites.

The syrup of the iodide of iron, in doses of 5 to 10 minims three times a day, often produces marked diminution of enlarged glands in the neck, and has proved useful in cases where the bronchial glands were also enlarged. Sulphide of calcium has been recommended in doses of one-twentieth to one-tenth of a grain every hour. Arsenic may be given in combination with iron, or with a vegetable bitter. Iodine in the form of the tincture, in doses of one to three minims, may be tried.

For the treatment of the cough and vomiting the writer advocates the employment of the glycerine of carboic acid, in doses of one to three minims, in combination with cod liver oil and iron. Creasote in capsules is also useful.

If complications arise, e.g. tuberculosis of the air-passages, erosion of the œsophagus, empyema, etc., they should be treated along the lines indicated under these headings.

Tracheotomy is only of use when a caseous mass is impacted in the trachea or glottis.

### Bronchitis

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See also ALCOHOL (*Clinical Uses*), ALCOHOLISM (*Complications*), ANESTHETICS, ETHER, ASTHMA, BRONCHI, BRONCHIAL GLANDS, BRONCHI, BRONCHIECTASIS, BRUIES AND SCABS (*Clinical Features, Respiratory Complications*), CHEST, CLINICAL INVESTIGATION OF CHILDREN, DEVELOPMENT OF (*Symptoms of Teething*), COUGH (*Clinical Varieties*), EXPECTORATION, GOUT (*Respiratory System*), HEART, MYOCARDIUM AND ENDOCARDIUM (*Symptomatology*), HYPERTROPHY, LUNG, TUBERCULOSIS OF, etc. MEASLES, NEPHRITIS, OXYGEN, PNEUMONIA, CLINICAL, PREGNANCY, AFFECTIONS AND COMPLICATIONS, RICKETS, SWELLING, THIRAPIEUTICS, HEALTH RESORTS, TRADES, DANGEROUS (*Cotton*), TUBERCULOSIS, TYPHOID FEVER (*Complications and Sequelæ*).

BRONCHITIS is an inflammation of the bronchial tubes, general or partial, involving either the larger or smaller bronchial tubes.

The term bronchitis is of comparatively recent date, having been introduced by Dr. Charles Bulham in 1810, when it superseded the old phrase "pulmonary catarrh" or "defluxion."

For the purpose of description the subject will be divided into the following sections:—1 Acute bronchitis in the adult, 2 Chronic bronchitis in the adult, 3 Bronchitis in children.

*General Considerations*.—It would naturally be supposed that the bronchial tubes, ramifying throughout the whole of the lungs as they do, should be so intrinsically a part of the lung that no clinical division could be drawn between inflammation of the bronchi and inflammation of the pulmonary tissue, pneumonia. And though from a clinical as well as from a pathological standpoint bronchitis is sharply differentiated from pneumonia so long as the larger and middle-sized tubes are alone affected, we shall seek in vain for any line of demarcation between capillary bronchitis and the lobular pneumonia with which, in children at any rate, it is almost invariably associated.

To appreciate the pathological changes taking place in bronchitis, it is necessary to bear in mind the normal structure and functions of the tissues involved. An explanation will be found for the tendency for the inflammation to be

mainly bronchial, firstly in the anatomical arrangement of the blood-supply, and secondly in the protecting action of the basement membrane, while even the marked variation in the clinical characters between bronchitis affecting the larger and smaller tubes very largely depends on their different anatomical structure and relations. The noteworthy point in regard to the vascular system of the lung consists in its double circulation, the blood-supply for nutrition and function being separately provided for, thus the main bronchi and the tissues of the lung are supplied, not by the pulmonary artery, but by the bronchial arteries which are derived from the aorta. Furthermore, the bronchial veins communicate freely with the pulmonary artery, and in the smaller bronchi the blood is returned almost wholly by branches of the pulmonary vein to the left side of the heart, consequently any obstruction to the return of blood to the left side of the heart will at once result in congestion of the smaller bronchi. Moreover, while it seems probable that the pulmonary arteries are not subject like most arteries to vasomotor control, this does not apply to the bronchial arteries, and thus a variety of causes acting either directly, or indirectly by reflex action, are able to cause a vascular dilatation which may be limited to the bronchial tubes, as distinguished from the an-veoles which are supplied by the capillary plexus of the pulmonary vessels, and which are directly concerned with the function of respiration—*aeration of the blood*.

The right and left bronchi divide and subdivide dichotomously within the lung until ultimately the minute terminal bronchioles end in the infundibula and an-veoles. In a transverse section of one of the middle-sized bronchi three coats may be distinguished—the inner mucosa, the middle muscular, and the outer fibrous coat, which contains the cartilages. Further examination shows that the mucosa is composed of (1) an epithelial lining with three layers of cells—the single layer of superficial columnar ciliated epithelium, an intermediary layer of transitional or rounded cells, actively germinating to form the superficial columnar cells and mucous corpuscles, and a single deep layer of flat nucleated cells, (2) an inner fibrous coat, abundantly supplied with branches from the bronchial artery, and consisting of bundles of fibrous tissue with numerous lymphatic spaces containing lymphatic corpuscles between them, and (3) a homogeneous, hyaline, structureless basement membrane giving attachment to the epithelial covering which it effectually separates from the vessels and lymphatic vessels of the inner fibrous coat. Hamilton has shown that the part played by this basement membrane in bronchitis is of great importance, in that it imparts a superficial character to the catarrhal affections of the bronchi, for though it becomes

swollen and oedematous in bronchitis, and permits the transudation of serous fluid into the lumen of the tube, no leucocytes appear to pass from the deeper structures to the surface of the mucous membrane, nor can inhaled particles of foreign matter, like coal-dust in a miner's lung, become carried down to the tissues of the bronchi. Such particles do not enter the lymphatics of the lung except through the an-veoles. In the middle-sized and smaller bronchi the muscular coat is well developed, and it is continued down to the infundibula, where it is represented only by a few scattered fibres. The physiological action of this muscle is not known exactly, but it probably regulates the intra-alveolar an-tension, and protects the an-veoles from undue pressure in coughing and other respiratory acts. Consequently, as Hamilton has demonstrated, this coat generally becomes greatly hypertrophied in chronic bronchitis, owing to the constantly recurring cough. Gardner, in discussing the action of expectorant medicines, arrives at the conclusion that expectoration depends more upon the expulsive mechanism of these muscular fibres than upon the alterations in the consistency of the secretions. Their action is probably somewhat analogous to intestinal peristalsis, and the beneficial result of emesis in certain cases of bronchitis is due to the remedies increasing the action of the bronchial muscles directly, rather than to the vomiting, which is perhaps only a concomitant, but in no way essential, effect. Further evidence of this important physiological action is afforded by the fact that, when the muscular coat loses its functional activity, as in the more serious cases of bronchitis, the bronchial tubes dilate and become filled with secretion. Walsh, referring to Radcliffe Hall's researches, considers that in the calm, and still more in the forced respiration of health, the muscular coat possesses the power of reducing the tubes to less than their medium size. But in bronchitis the muscular coat loses its contractile force, the elasticity of the inflamed bronchi being likewise impaired, and so become intelligible the tendency to dilatation of the tubes, the laboured expiration and the accumulation of mucus.

It is not improbable that, like the larynx and vocal cords, the bronchial muscle may rhythmically dilate and contract with inspiration and expiration. I believe that this largely explains why in bronchitis and asthma the dyspnoea is expiratory rather than inspiratory, inasmuch as the muscular spasm is more or less inhibited during inspiration, permitting air to be inspired more freely than during the period of bronchial spasm it can be expired, consequently the lungs become distended and emphysematous. In the larger and middle-sized bronchi the outer fibrous coat contains cartilaginous plates, but not in the smallest bronchi, which consist of a

layer of stratified ciliated epithelium, surrounded by the muscularis and peribronchial connective tissue. If one of the bronchioles be followed towards its termination, it is found that just before it ends in the infundibulum the columnar ciliated epithelium is replaced by a single layer of cubical epithelium, a thin fibrous tissue investment being still present, and finally when the air-vesicle is reached this epithelial layer comes to be composed of a very thin layer of flat, squamous, endothelial-like cells, while the muscular coat is lost.

The thinness and weakness of the walls of the smallest bronchi, and the feeble expulsive power of their muscular coat, together with the narrowness of the lumen of the tube, and especially the complete absence of ciliated epithelium, and of any muscular coat as they approach the infundibula, explain the serious nature of a capillary bronchitis and the danger resulting from secretions accumulating in the smallest tubes.

#### 1. ACUTE BRONCHITIS IN ADULTS

*Etiology*.—We may divide the causes of acute bronchitis as follows:

(a) *Predisposing*.—No age is exempt, but it is especially liable to attack the very old or the very young, and to assume a serious form in these subjects. Males and females are equally liable to suffer. Sedentary life and luxurious habits, and living much indoors and in vitiated atmosphere, heart-disease, especially those forms which result in pulmonary congestion, and gout, diabetes, rickets, dyspepsia and constipation, spinal curvature, dusty occupations, and a cold and changeable climate, all predispose to bronchitis. But heredity and a certain individual susceptibility to catarrhal affections undoubtedly largely influence the occurrence of attacks, and, above all, the pre-existence of various pulmonary affections and the fact of previous attacks of bronchitis having taken place.

#### (b) *Erecting causes*

1 Exposure to cold, "catching cold."

2 Traumatic, the inhalation of steam swallowing hot water or irritating fluids.

3 Chemical irritants, e.g. the inhalation of irritating gases or vapours such as chlorine or ammonia, sulphurous or nitrous acid, ether, iodine, bromine, etc.

4 Toxicæmia, e.g. in Bright's disease, gout, rheumatism.

5 Vasomotor, e.g. asthma.

6 Infective, e.g. typhoid, typhus, measles, scarlatina, smallpox, malaria, whooping-cough.

Acute bronchitis is generally associated with exposure to cold, and it is certainly influenced by climatic conditions. But although a so-called chill is undoubtedly a very common exciting cause of bronchitis, it is equally certain that in many cases the real cause of the attack is by no means so simple. Indeed we find that it is not those whose daily life constantly exposes

them to cold, or even rapid changes of temperature, that are most prone to attacks of bronchitis, "persons who live an out-of-door life are usually less subject to the disease than those who follow sedentary occupation" (Osler). Certain it is that colds are not usually caught in pure air, at sea for instance, or in the higher Alps in winter, although exposure to cold is of necessity frequent. Acute inflammatory attacks of the upper air passages are most frequently found in those who habitually breathe vitiated air, or who have attended over-crowded, ill-ventilated places of meeting, and are then exposed to cold, or who are directly infected by some person suffering from a cold in the head. Not only do these "catarrhs" confer temporary immunity, but recent investigations, such as those of W. H. Park, have shown that staphylococci and streptococci, which are always present in the healthy mouth, increase greatly in number and violence in damp weather and in winter months, and are then capable of setting up acute pharyngitis if applied to the throat. These are just the conditions which are particularly prone to lead to bronchitis, and we thus seem to have an explanation of its frequent occurrence in the changeable weather of early spring and late autumn, and of its origin in a "cold on the chest."

Sputum normally contains a considerable number of different varieties of micro-organisms, some of which are best studied on stained films, whilst others grow well on the ordinary media. Amongst the most common of these organisms are the streptococcus brevis and other varieties, staphylococcus albus, citreus, and aureus, spillum spitzogenum, leptothrix, and varieties of chromogenic bacteria, bacilli and sarcine. Whilst any one of these may be found in the sputa of bronchitis and bronchopneumonia, it will commonly be found that one or more organisms are present in overwhelming majority, so that prepared films or cultures may show almost a pure culture of one particular variety. Pausani found in normal air-passages several different streptococci, bacilli, micrococci, sarcine. Queyral describes a specific organism, a micrococcus of "tracheo-bronchitis." Grün describes a specific bacillus of bronchitis, but neither of these observations has been substantiated. Netter gives the pneumococcus as the cause of capillary bronchitis and broncho-pneumonia in 15.85 per cent of cases.

Osler makes the statement that acute bronchitis is probably a microbial affection, and Gee, in referring to the effect of dust in certain occupations in causing bronchitis, states that the dust of unclean and ill-ventilated rooms will be very likely to contain moribund microbes, and that the irritation of the air-passages set up by inanimated dust will favour the operations of the many bacteria which are potent to cause bronchitis. He believes that our mouths, noses, and

throats harbour many morbid microbes in a latent state, there awaiting a favourable opportunity for becoming active and virulent, and thus setting up the catarrhal attack which spreads down to the trachea and bronchi.

No doubt many of the cases of tracheo-bronchitis originate in this manner; yet, notwithstanding the bacteriological researches which have been just mentioned, I am convinced that in a larger proportion of the cases of bronchitis in which the middle-sized bronchi are mainly implicated from the outset, exposure to cold is itself the immediate cause of the disease. It may be brought about directly by cold air entering through the upper air passages, as shown, for instance, by the experience of susceptible persons on going from a heated atmosphere into cold night air, by the liability to bronchitis in tracheotomised patients, or in habitual mouth-breathers owing to the inspired air not being duly warmed by passing through the nose. But in most cases it seems that the action of cold is indirect, and the result of vasomotor paralysis, as explained by Rosenthal; for often enough the cause of the attack has been getting wet, or lying on damp grass, or sitting still after getting over-heated and perspiring, and not by the action of cold air entering through the mouth.

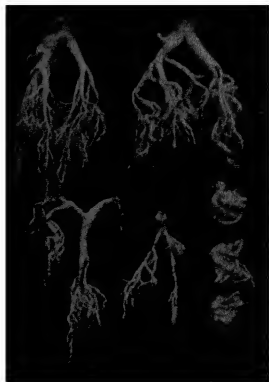
Other deep-seated viscera having no air-containing tubes of communication to the surface of the body, such as the bladder and kidney, are similarly and specially subject to inflammatory attacks from exposure to wet and cold applied to other parts of the body; and in applying hot fomentations or poultices to the chest, to the hypogastrium or lumbar regions in inflammations affecting respectively the lungs, bladder, or kidneys, we take advantage of these associated reflex vasomotor areas.

We may further refer to the secondary bronchitis which is liable to arise in the course of influenza, typhoid fever, measles, and other exanthemata. It is widely recognised that the bronchitis of influenza, which is certainly microbial, is especially liable to be aggravated by the least exposure to cold, while the pulmonary complications of measles are particularly prone to be severe if the rooms are at all crowded or ill ventilated. It is a remarkable fact that influenza epidemics are just as prevalent at Davos and St. Moritz as in the less dry atmosphere of England and Scotland, and that, despite the ideal climate of the higher Alps for most bronchitic affections, cases of influenza at these Swiss resorts are just as prone to be complicated with severe bronchitis and pneumonia. Thus a chill, while undoubtedly capable of directly causing acute bronchitis in those unaccustomed to exposure, or in those who seem to have a special liability to catarrhal affections of the respiratory tract, is often an indirect rather than a direct influence in causing the

disease, and there are the other causes of bronchitis which can in no way be associated with either cold or micro-organisms, as the inhalation of irritant vapours such as strong ammonia, chlorine gas, etc.

*MORBID ANATOMY.* The morbid changes underlying bronchitis in its various forms are primarily an exaggeration of the normal physiological processes taking place in a healthy lung, and we shall find that inflammation of the bronchial tubes is essentially similar to inflammation in other mucous membranes, with certain modifications due to their peculiar anatomical structure and relations, so that the course of the disease, and the particular clinical type it assumes in different cases, vary not only with the actual cause or the acuteness of the attack, but depend in a large measure on the calibre of the bronchial tubes most concerned.

It is not very often that we are afforded an opportunity of examining post-mortem the actual conditions presented by any but the directly fatal and therefore most severe forms of bronchitis. Socoleff, who induced bronchitis in animals by the application of chemical irritants,



and Hamilton, from post-mortem observations on patients dying with bronchitis, have investigated the histological changes which occur in the bronchi in health and disease, and I am mainly indebted to the writings of the latter for the following description of the morbid anatomy of bronchitis.

The initial change is hyperæmia, followed by swelling of the mucous membrane and excessive secretion. The hyperæmia, as Biernier demonstrated, may first affect either the superficial or deeper structures, or both.

According to Hamilton, the earliest departure from the normal condition is a relaxation and distension of the attendant plexus of blood-vessels

ramifying in the inner fibrous coat, immediately beneath the basement membrane—that is to say, of the branches of the bronchial artery. In a few hours the basement membrane became swollen from serous infiltration. Next, and quite early in the course of the affection, Socoleff and Hamilton found that the ciliated epithelial layer desquamates, and is not regenerated till recovery takes place, when it is gradually reproduced. The cells of this layer undergo fatty degeneration, and Hamilton says that it is no doubt partly destroyed by this means and partly exfoliated, while others may be inhaled into the smaller bronchi, where they may be seen lying in large detached masses among the other catarrhal products. Wilson Fox and Biernier consider that the epithelial desquamation is largely a post mortem change, and Hamilton admits that the shedding is seldom complete, the deep layer of transitional cells, or at least the single layer of flat germinating cells, usually remaining, and by a process of histoplastic division rapidly reproduces the nucleated cells, which, being continually thrown off, give to the secretion its peculiar catarrhal character. At the same time the mucous glands are particularly active, and it is the combination of this catarrhal mucus with the epithelial cells which gives rise to the so-called muco-purulent fluid which occupies the bronchial lumen until removed by expectoration. After about the tenth day, Hamilton found that the congestion not only affects the inner fibrous coat, but that all parts of the bronchus exhibit vessels over-distended and engorged with blood, and that, *pari passu* with the changes in the epithelial covering, the inner fibrous coat becomes infiltrated with cells derived from the endothelium lining the lymphatic spaces, until the whole of the lymph-spaces become choked by these new cellular products. These cells, according to Hamilton, never extend towards the free surface of the mucous membrane, the basement membrane forming an impassable barrier in that direction, but they invariably spread outwards, through the inter-muscular lymphatic spaces, to the adventitia, where similar changes will be found to have taken place. But this proliferation, with the production of leucocytes, soon spreads throughout the lobular septa to the deeper layers of the pleura, the whole of the lymphatic vessels becoming the subject of a catarrh, while the lymphatic glands at the root of the lung are then invariably found enlarged.

The hyperæmia of the mucous membrane may almost entirely disappear after death, but as a rule it is bright red, or in cases of longer duration dark purple, and presents a thickened, opaque, and velvety appearance, the lumen of the smaller tubes being diminished by these changes, and filled with thick, opaque, yellow secretion which exudes from the small bronchi from the cut surface of the lung on pressure. Bronchitis

of the smaller tubes is accompanied by pulmonary congestion and œdema, and generally areas of collapsed lung or foci of lobular pneumonia may be found.

Again, Hamilton has found that the first indication of recovery taking place in bronchitis seems to be the diminution of the congestion of the mucous membrane, the vessels apparently recovering their tone, the proliferation of the epithelium becoming less active, the cells being once more fully developed into columnar cells, while the cellular infiltration of the bronchial wall and the lymphatic vessels becomes gradually absorbed in cases which proceed to recovery.

In those cases which, from the severity of the attack, or from failure of the factors which make for recovery, or which from the outset owing to the persistence of less acute causes, pass into the condition of chronic bronchitis, the mucous membrane and entire bronchial wall remain thickened, the muscular coat becomes hypertrophied, while the mucous glands and the bronchial cartilages more or less completely disappear, being replaced by dense cellular infiltration. In advanced cases, however, the muscular and the adventitia become atrophied probably owing to the cellular infiltration of the lymphatics, and thus arise those changes which from various causes result in bronchiectasis, emphysema, interstitial pneumonia, and other pulmonary complications which are beyond the scope of this article.

*Collapse of the Lung*.—A collection of the catarrhal secretion of bronchitis may temporarily occlude a smaller bronchus, resulting in weakness or absence of the vesicular murmur over the corresponding portion of the lung. On the patient coughing, the occluding mass of secretion may be dislodged, the air again entering the air-vessels. But the inflammation may extend to the smallest bronchioles, or the mucopurulent secretion may be sucked down so as to plug a bronchial channel which itself is unaffected. Especially if in any part of the lung several of these finer divisions are occluded, no effort of coughing may suffice to dislodge it. The air in the corresponding portion of the lung then becomes absorbed by the surrounding pulmonary capillaries, and the air-vesicles collapse, with more or less so-called "compensatory emphysema" in the other portion of the lung. Collapsed lung has a pinkish red appearance, and on section is reddish brown, giving a frog-spawn sensation to the touch, and the corresponding surface of the lung is depressed. Merely collapsed lung does not as a rule become inflamed, unless it be from extension of inflammation from a neighbouring patch of pneumonia, and on the removal of the obstructing cause, if this be not too long delayed, may again become inflated. But coincidently with the occurrence of collapse, patches of lobular pneumonia, with true red hepatization, are



usually found, probably due either to extension of the inflammatory process right down to the air-vesicles and pulmonary stroma, or to direct infection by various micro-organisms. (For fuller details see "Pneumonia.")

**Symptoms.**—Acute bronchitis in its clinical aspects presents every gradation from a mild and transient cold on the chest to an intensely acute and rapidly fatal disease attended with urgent dyspnoea, cyanosis, and collapse. In the milder forms, or tracheo-bronchitis, the attack is usually ushered in by the symptoms of an ordinary "cold," with lachrymation, sneezing, and a stiffness and soreness of the throat. The catarrh may extend gradually to the chest, involving the larynx with resulting hoarseness, and causing a sense of rawness and soreness behind the sternum. In other cases the cold seems to settle on the chest from the outset without obvious pharyngitis and laryngitis. Initial rigors are not usual, but a sense of chilliness, shiverings, pains in the bones and back, general malaise, headache, drowsiness, and languor, with more or less gastric catarrh, hepatic distensions, and constipation, commonly characterise the earlier stages. The pulse and respiration are moderately quickened, the skin, at first dry, soon becomes moist.

In still other cases, particularly in the old or very young, the onset may be almost sudden, with severe cough and urgent dyspnoea, as Wilson Fox describes it, almost reproducing the phenomena of spasmodic asthma, but differing from the latter in its persistence, or cough and dyspnoea may set in acutely, but without so distinct spasmodic element, followed after a few hours by the expectoration of an abundant, glairy, blood-stained mucus.

In milder cases the temperature is but slightly raised, but with the more severe it may range between 101° F and 102° or 103°, showing the usual evening rise and the morning decline.

The urine is of the usual febrile character, of high specific gravity, high-coloured, loaded with lithates and mæa, and free from albumen unless complications are present.

The cough at first is harsh, loud, and ringing, either coming in frequently repeated single coughs, or in paroxysms which are exceedingly harassing to the patient, accompanied by a sense of oppression and tightness in the chest and a considerable soreness beneath the sternum and along the attachments of the diaphragm, the severe paroxysms sometimes ending in vomiting. It is often most distressing in the earliest stages before there is any expectoration, being then due to irritation of the inflamed bronchial mucous membrane, the irritation being referred to the laryngo-tracheal region. After an interval the cough is attended with expectoration, which at first is thin and watery, and frothy, consisting of serous exudation containing the ciliated epithelium which is shed

very early. As the disease progresses, it becomes more consistent andropy, from the increased exudation of mucus, and more or less opaque, owing to the numerous round cells thrown off from the deeper epithelial layers, becoming in turn muco-purulent and purulent. The cough is then due, not to irritation of the dry mucosa, but to the accumulating secretions which require to be got rid of, and is therefore apt to be more severe on rising from the recumbent position after sleep. Streaks of blood are sometimes seen in the sputum, especially in the earlier stages when the cough is severe.

Dyspnoea is seldom pronounced when the larger tubes alone are affected. When the smaller tubes are extensively implicated, owing to the excessive contraction of the muscular coat acting in the manner explained above, and perhaps in part from the valve-like action of the accumulating secretion, more air is drawn into the lung than can be expired, and the lungs consequently become over-distended and the respiratory movements shallow and ineffectual. It is remarkable that the dyspnoea is abdominal and expiratory, rather than inspiratory, in contradistinction to the dyspnoea due to obstruction in the larynx or trachea. When the air-vesicles are already distended with the air which no effort of the patient can effectually expire, but little or no more air can enter, and thus there is not much tendency for the secretion to be sucked in. Expectoration is then attended with much difficulty, and the thick, yellow, sticky secretion from the small tubes is seen to hang in strings suspended in the lower watery layer from the upper layer of frothy mucus from the upper larger bronchi. Sometimes the expectoration is very scanty in capillary bronchitis, even when moist râles are audible over the chest.

An attack of acute bronchitis may assume an asphyxiating type, either from a simple bronchitis of the larger tubes occurring in a debilitated patient, especially in the emphysematous, or from a very extensive implication of the smaller bronchi, or from widespread involvement of the capillary bronchi. Though capillary bronchitis in the adult may be remarkably sudden and severe in its onset, it not infrequently commences with the symptoms of an ordinary mild bronchitic attack. Rigors are rare, says Walshe, and vomiting rarer. But nervous symptoms supervene early and are pronounced, the patient is restless and drowsy, the pulse rapid and small, cyanosis is a marked feature, and there is complete loss of appetite. Muttering delirium at night and sleeplessness are common, but acute maniacal excitement is not unknown. The acute dyspnoea, cyanosis, and active, sometimes maniacal, delirium when strong, young adults are the subject of acute capillary bronchitis, form a very painful clinical picture. If the attack cannot be relieved the patient rapidly becomes asphyxiated. Walshe,

in his description of this type of bronchitis, states that, "as long as the strength permits, the patient sits or bends forwards, but the body gradually yields, and it is not uncommon to find patients, while still perfectly conscious, lying sideways or forwards, with the head lower than the shoulders, and in rare cases this posture of the head is adopted from the very outset."

**Physical Signs.**—The chest may not be altered in shape in mild cases, but in pronounced attacks assumes the inspiratory type, it remains more or less fully expanded, the respiratory movements being elevatory rather than attended with expansion and contraction of the parietes, and are violent, and increased in frequency, though not in amplitude. The abdominal movements are increased, and if the bronchial tubes are extensively involved, there will be marked cyanosis and fullness of the large veins of the neck. The right side of the heart dilates, giving rise to epigastric pulsation, or the heart may be pushed down, the impulse being felt in the left costal angle. The liver is often congested and displaced downward. But in the acute asphyxiating type, as has already been stated, the respiratory movements of the over-distended chest are increasingly shallow, and the heart's action is apt to fail rapidly, with marked cyanosis, clammy perspiration, coldness of the skin, general anasarca, and scanty albuminous urine.

The adventitious sounds which arise in bronchitis are of two kinds—the dry (rhonchus and sibilus) and the moist (râles). Rhonchus, or sonorous rhonchus, is a loud, deep-toned, cooing sound, due to air passing through one of the larger tubes which is partly obstructed by a collection of mucus, producing a *vine fluide*. The mucus can often be removed by the patient coughing, hence rhonchus audible at one moment may abruptly disappear. Sibilant rhonchi, or sibilus, is a more high-pitched whistling sound, due to swelling of the mucosa in the small bronchi, hence they are of greater importance, and as they cannot be dispelled by coughing, they tend to persist in the same region for a considerable period.

A rhythmic rhonchus or sibilus, as was first observed by Stokes, may be produced by the ventricular contractions, if the portion of lung is in close proximity to the heart and the secretions abundant.

Râles may be large, medium, or small, according to the size of the bronchial tube which is occluded by an accumulation of mucus, the sound being produced by the air passing through the mucus. Moderately fine râles, however, may be produced in the larger bronchi. Large râles are of less grave import than the small, in that they imply that the larger tubes are mainly affected, and mere loudness of the sounds is not of bad omen—in fact, it is in the graver cases of widespread

implication of the finer tubes that the moist sounds are least audible. The smaller râles are sometimes spoken of as sub-crepitant. Yet it is of considerable importance that they should be distinguished from the crepitation which is indicative of pneumonia or of phthisis, conditions which may be associated with the symptoms of simple bronchitis.

A plug of mucus in a bronchus may temporarily prevent the air entering the corresponding portion of the lung, and causes a localised diminution or absence of breath-sounds, until by coughing the plug is removed. But usually in bronchitis the amount of air entering the vesicles being deficient, the normal vesicular murmur is less distinct than in health, consequently the bronchial breath-sounds are less marked and the breathing seems unduly harsh. Yet the bronchial breathing is never heard in uncomplicated bronchitis, except over the roots of lungs, it is only heard when either collapse or pneumonia is present, and may then be associated with dullness on percussion and increased vocal resonance.

Vocal fremitus and voice-conduction are scarcely altered in uncomplicated bronchitis.

The pectoral-note is normal unless the bronchitis is pronounced, and then the whole of the upper part of the chest, being acutely distended and emphysematous, yields a hyper-resonant or almost tympanic note. Even small patches of broncho-pneumonia do not give rise to dullness on percussion, but congestion of the bases, collapse, or pneumonia, if extensive, causes a diminution of the resonance. The tongue is almost invariably coated, and symptoms of gastro-intestinal catarrh, with anorexia, thirst, and constipation, are generally present.

In uncomplicated cases of the larger and middle-sized tubes the attack gradually subsides in the course of a week, the expectoration becoming decreased in amount and more normal in character, the physical signs in the chest gradually disappearing, convalescence being established in about ten days or a fortnight. But in the old and feeble, and in young children, the disease is apt to extend down to the smaller tubes, with a liability to pulmonary collapse. Especially is this dangerous complication likely to arise in measles and whooping-cough. In the acute suffocative type the prognosis is always grave, even in cases uncomplicated with cardiac or renal disease, while any coexistent affection of the lungs, heart, or kidney greatly aggravates the danger.

Walsh said he had known a case fatal in forty-six hours, but while these patients often succumb within a few days, they sometimes recover even after the condition appears hopeless.

Acute bronchitis may pass into a subacute or chronic state, and is then liable to be associated with various complications.

**DIAGNOSIS**—The diagnosis of acute bronchitis rarely presents any difficulty. Though it may be abrupt in onset, and even attended with considerable fever, there is not that initial rigor and characteristic disturbance in ratio between respiration and pulse-rate of pneumonia, while the absence of fine crepitation or tubular breathing, the normal and exaggerated resonance of the chest, and the widespread presence of coarse râles and rhonchi are sufficient to differentiate bronchitis from pneumonia.

Acute pulmonary tuberculosis may closely simulate, and for a time be indistinguishable from, acute bronchitis. High pyrexia and great prostration with early delirium, while not excluding simple bronchitis, suggest acute tuberculosis, the occurrence of haemorrhages may assist the diagnosis in doubtful cases (Wilson Fox).

It is, however, necessary to bear in mind that bronchitis may be pronounced in the early stages of typhoid fever, measles, whooping-cough, and more rarely variola, scarlatina, and other affections, the indications of which must not be overlooked. Thus the initial lachrymation, conjunctival injection, sneezing, and rhinorrhœa would lead to the suspicion of measles. Headache, general malaise, the peculiar rise of temperature, tenderness and gurgling in the right iliac region, enlargement of the spleen, the characteristic rose-spots and other indications of typhoid fever should be eliminated. The aching in the lumbar region in variola and the sore throat of scarlatina and early appearance of the rash seldom leave one in doubt for any considerable period. Whooping cough may closely simulate simple acute bronchitis, and, prior to the development of the characteristic crowing, may be indistinguishable.

**TREATMENT**—In the milder form of bronchitis of the upper bronchial tract—tracheo-bronchitis—domestic remedies for a cold on the chest usually suffice, and these only call for mention. The mustard and hot-water foot-bath, the application to the chest of a mustard poultice, or hot fomentations sprinkled with turpentine, followed by a Dover powder, and an aperient, and some simple diaphoretic mixture such as sweet spirits of nitre, and small doses of ipecacuanha, will often prove sufficient to relieve and shorten the attack. If the patient cannot remain indoors for a day or two, it is necessary to avoid diaphoretic remedies, which render him more susceptible to cold, and involve a risk of converting a slight bronchial attack into the much graver general bronchitis of the smaller tubes, and for this reason a Turkish bath in the initial stages of bronchitis is attended with a new risk.

In severer attacks more active measures are needful, and it should be ascertained whether the bronchitis is a primary affection, or secondary

to some constitutional condition or other disease, e.g. Bright's disease, which would call for appropriate measures.

In acute primary bronchitis the patient should be confined to his bed, and the temperature of the room maintained at about 62° to 65° F. We have seen that the first stage is due to vascular enervation and engorgement of the mucous membrane. Thus the chief indication is to combat the dry and congested condition by improving vascular tone and inducing free secretion.

At the outset a large mustard and linseed-meal poultice should be applied to the front and back of the chest, this being replaced by simple linseed poultices, after the mustard has sufficiently stimulated the skin to produce thorough redness. A steam-inhaler, with compound tincture of benzoin or tincture of belladonna added to the hot water, often affords relief. A moist atmosphere is not only comforting to the patient, but seems to materially relieve the dry, hacking cough, but when a steam-kettle is used, it must be remembered that the steam may condense on the bed-clothes, and special care is needful, by ensuring the maintenance of the temperature of the apartment, to obviate the risk of a chilling effect on the patient. During the early period in the attack the drugs that are most relied on are tartarised antimony and ipecacuanha, apomorphia, or squills, to promote bronchial secretion, together with citrate of potash, or acetate of ammonia, spirits of nitre, or some other diaphoretic. If there is bronchial spasm, tincture of lobelia or belladonna, citrate of caffeine, chloral, or opium may be added, but should be cautiously used, especially in children and in the old or feeble.

As soon as the dry stage passes off and secretion commences, antimony and the diaphoretic remedies may be discontinued, and stimulant expectorants, such as carbonate of ammonia with senega, squills, euphorbia pulifera, or small doses of iodide of potassium, substituted. At this stage the preparations of opium and direct sedatives to check the cough which is due to the accumulation of secretion in the tubes should be avoided.

When the smaller tubes are extensively involved and the amount of secretion is very considerable, the patient must be carefully watched for any indication of cardiac failure or pulmonary collapse. In weakly patients with impaired heart-action it may be necessary to resort at once to alcoholic and other stimulants, such as digitalis, strychnine, or ether.

If the bronchial secretions tend to collect in the smaller tubes and pulmonary collapse is threatening, it may be desirable to induce vomiting by the administration of sulphate of zinc or by repeated large doses of ipecacuanha. Depressing emetics should be avoided.

With threatening apnoea, the use of oxygen inhalations, or, on rare occasions, venesection, may be indicated. It is hardly necessary here to give directions in regard to diet in bronchitis, beyond emphasising the importance of keeping the patient on low diet in the beginning of an attack, for the gastro-intestinal tract is always more or less deranged, and calls for appropriate treatment.

## II CHRONIC BRONCHITIS IN ADULTS

**ETIOLOGY.**—*Chronic bronchitis* is due to much the same, though generally more persistent, causes as the acute form, but the influence of predisposing factors and constitutional conditions is more potent, and is more usually the essential cause of the chronic affection. Moreover, chronic bronchitis is distinguished rather by its protracted course and secondary complications than by any special clinical features. In some cases frequent subacute attacks, by increasing the susceptibility of the individual, and resulting in a gradual permanent impairment of the tissues, pass insensibly and without any line of demarcation into the chronic form. Thus chronic bronchitis may follow repeated attacks of acute bronchitis, or may be chronic from the beginning. It is usually met with in the aged, the "winter cough" of old people recurring from year to year throughout the colder months. Various diatheses, such as syphilis, gout, and renal disease, heart affections, especially disease of the mitral valve, aneurysm, and chronic lung affections, such as phthisis, emphysema, and dilated bronchi, and almost any condition impairing general health, predispose to chronic bronchitis. But a mild form is also met with in children and young adults who suffer periodically from recurrent catarrhal attacks, either on the slightest exposure, or it may be associated with gastro-intestinal disturbance. Chronic bronchitis is also very common in workers in dusty occupations, mill-puff upholsterers, bakers, colliers, etc., and is often associated with chronic alcoholism.

**MORBID ANATOMY.**—The mucous membrane of the bronchi in chronic bronchitis is smooth, slimy, and of a slaty grey or dark purple colour, a brighter colour being often present in post-mortem examinations, due to an acute exacerbation which so often causes the fatal result. Close examination will show the open mouths of the dilated mucous glands like pin-point depressions. The mucous membrane is often thrown into folds by the accumulation of cellular structures beneath the basement membrane, and the epithelium consists either of the single layer of flat germinating cells or heaped-up collections of transitional cells, fully formed ciliated epithelium being generally absent. The smaller bronchi are filled with sticky yellow muco-purulent secretion which oozes from the open mouths of the tubes on

section of the lung. In uncomplicated chronic bronchitis the muscular coat is hypertrophied, and the tunica adventitia thickened from cellular infiltration, which also extends to the peribronchial and peri-vascular fibrous tissue. The cartilages are usually atrophied, or have disappeared entirely. In old patients they may undergo calcareous degeneration. The lungs are usually emphysematous, and in very chronic cases with dilatation of the bronchi the muscular coat may have disappeared. The mucous membrane may be ulcerated in fatal bronchitis and in bronchiectasis. The lymphatic glands are invariably enlarged and pigmented, and frequently caseating. The heart, and especially the right ventricle, is generally dilated, and the liver, spleen, and kidneys are chronically congested, and the seat of fibroid degeneration.

**SYMPTOMS.**—The symptoms vary considerably in different cases, but mainly resemble in character those of the acute form, especially modified by coexistent affections. In the milder forms of the common winter cough of old people at first the only complaint is cough with mucopurulent expectoration and slight dyspnoea on exertion, or in the early morning, unless there is marked coexistent emphysema. There is no pain or fever in the earlier stages. With the return of warmer weather the attack passes off completely, only to return with the recurrence of cold and changeable climate. The affection may continue in this manner for several years, but gradually dyspnoea on the slightest exertion is noticed as the attacks increase in severity, and the cough, instead of disappearing during the summer months, tends to persist all the year round, the incessant cough and purulent expectoration disturbing the patient's sleep, the patient's health and strength being gradually undermined.

In the young and in healthy adults the milder cases generally subside gradually with complete convalescence, but when once chronic bronchitis has become pronounced and well established, the pathological structural changes which arise render complete recovery scarcely possible.

**PHYSICAL SIGNS.**—The chest may be moderately distended without alteration in shape, but patients with old-standing chronic bronchitis are almost invariably emphysematous, in which case the chest is considerably distended and barrel-shaped, the respiratory movements are limited, expiration being prolonged, the percussion-note is hyperresonant and clear, the breath-sounds are harsh and loud, deep-toned rhonchi, high-pitched squealing, piping sibilus, large and small râles, or bubbling, according to the varying conditions of the tubes, are constantly to be heard, while finer crepitation may often be found at the bases.

The expectoration is very variable in quantity, being muco-purulent, and sometimes streaked

with blood from the rupture of small bronchial vessels

**COURSE AND EVENT**—In the milder cases the general health may not be greatly impaired, but in course of time, especially when the lungs have become emphysematous, the heart becomes dilated, and evidence of cardiac failure is shown by chronic gastric catarrh, enlargement of the liver, and in some cases by albuminous urine. These secondary complications are no doubt largely due to the affections which are the real cause of the bronchitis. Various other pulmonary complications, such as bronchiectasis and chronic interstitial pneumonia, are liable to occur, and the patient sooner or later succumbs to a general failure of health and strength, unless an acute exacerbation, which is very liable to arise, or some intercurrent affection, carries him off.

**DIAGNOSIS**—The cough and expectoration without consolidation of the lung usually leaves no doubt as to the diagnosis. From pleurisy it is distinguished by the persistence of vocal fremitus and resonance, and respiratory murmur, and by the absence of bulging, and by the presence of the râles, from pneumonic consolidation, by the absence of complete dullness, tubular breathing, and bronchophony.

The conditions which are most liable to cause difficulty in diagnosis are tuberculous deposits, and bronchitis due to the presence of aneurysm or of new growths pressing on the bronchi.

**PROGNOSIS**—The prognosis depends mainly on the age of the patient and the coexistence of complications. Once established, it is only in the comparatively young and robust that complete recovery can be hoped for. In older patients the presence of well-marked emphysema, bronchiectasis, or any valvular heart affection must add to the dangers of intercurrent affections or acute exacerbations.

#### CLINICAL VARIETIES

The condition of the muscle of the right heart is of even greater import than the integrity of the cardiac valves, for the latter defect is often largely discounted by compensatory hypertrophy, whereas a degenerated and weak cardiac muscle is unable to cope with the demand for increased energy which is made on the right heart in bronchitis, and often fails to respond to any treatment.

The above description applies to the great majority of cases of varying severity. Attention has already been directed to the clinical importance of various diatheses and diseases which predispose to bronchitis. Often enough the bronchitis is only a prominent symptom of some such underlying disease, and some writers distinguish various clinical types, such as renal, gouty, syphilitic bronchitis, and so forth.

But there are certain forms of chronic bronchitis which call for special mention, viz.

(a) *Bronchorrhœa*, characterised by excessive secretion of glairy, semi-transparent matter, like white of egg mixed with water, containing greyish or yellowish-green masses, or the expectoration may be thin, watery, and clear—*bronchorrhœa serosa*. The cough and dyspnoea are usually paroxysmal, either limited to an hour or two on awakening in the morning, or coming on at intervals of several hours during the day, the amount expectorated being very large, sometimes as much as three or four quarts in the course of the day. During the paroxysms, dyspnoea is urgent, but in the intervals, and in fact throughout the day in some cases where the cough and expectoration only occur on waking in the morning, dyspnoea may be absent or scarcely noticeable. Râles and rhonchi are audible before and during the periods of expectoration, but in the intervals, until the secretion has re-accumulated, the auscultation may reveal comparatively few adventitious sounds. These cases are almost invariably associated with some degree of bronchial dilatation.

Patients may continue to live with little alteration in their condition for many years, but gradually the symptoms become more pronounced, the dyspnoea increases with a tendency to asthmatic symptoms, and generally failure of the circulation, increasing œdema, cyanosis, and impairment of health and strength.

(b) *Dry (Chronic) Bronchitis*.—The catarrhæ seræ of Laennec is characterised by paroxysms of very troublesome and severe cough, with very scanty expectoration of small masses of tough, viscid, hyaline mucus. The affection is almost always complicated by emphysema, and is usually associated with gout. It is generally regarded as due to congestion of the tubes, but "bronchial spasm is doubtless largely associated with the congestion, indeed, bronchial susceptibility and bronchial irritation are its unmistakable etiological factors" (Ewart).

(c) *Fœtid Bronchitis*.—In the course of a long-standing chronic bronchitis, the expectoration occasionally assumes almost suddenly, and without apparent cause, a dirty grey colour with a peculiar putrid odour. Putrid expectoration is met with in bronchiectasis, gangrene, and other destructive lesions of the lung, and in perforating empyema, and is usually due to one or other of these affections, but rarely it supervenes in uncomplicated cases of bronchitis. Its onset is generally attended with rigors, and with all the appearance of the occurrence of acute bronchitis superadded to the chronic affection, with fever of typhoid character, and attended with intense depression. The expectoration consists of a greyish-white alkaline, putrid, muco-purulent fluid, with a peculiar, sickly, characteristic odour which is said to suggest the smell of acacia blossoms. The amount secreted is very considerable, the sputa

separate into an upper fluid layer covered with froth, and a lower dirty layer containing yellowish plugs varying in size from a millet-seed to a bean, "Dittich's plugs." These masses on examination are found to be made up of pus-cells, oil-globules, fatty acids, leucin and tyrosin, and detritus. Various micro-organisms have been isolated from the plugs, *leptothrix pulmonalis* (Leyden and Jaffé), a short slightly curved bacillus which on culture gives an odour like that of the sputum (Lummizer), and short thick rods resembling *bacillus coli* (Hitzig). Vuchow and Gangee observed that these masses stain blue with iodine.

In the milder forms when the patient's strength and general condition is good, the prognosis is fairly favourable, but in severe cases it is liable to lead to various dangerous complications, such as pneumonia, bronchiectasis, gangrene, etc., with usually a fatal result. Death has occurred in some cases from the formation of metastatic brain abscess, but more usually the patients succumb to a general depression and collapse.

(d) *Plastic Bronchitis*.—The special feature of this peculiar and rare form of bronchitis is the expectoration of branching casts of the smaller bronchial tubes. Somewhat similar casts or moulds of the bronchi may be found by extension downwards of membranous laryngitis, and are occasionally found in pneumonia, phthisis, erysipelas, and other diseases, or as the result of severe irritation by the inhalation of steam, ammonia, etc., and fibrinous blood casts may be expectorated in hemoptysis. From all these conditions true plastic bronchitis differs in pathology and symptoms.

*Etiology*.—The cause of the affection is not known, and it is possible that in different cases the etiology is not identical. Its appearance seems to be due to some idiosyncrasy or peculiar features on the part of the sufferer. It has often been associated with a predisposition to tuberculosis, but it may attack those apparently in robust health. The exciting causes of the attacks are much the same as in ordinary bronchitis, generally occurring in the early spring months and after exposure to cold. It is nearly twice as common in males as in females, and, while it may be observed at all ages, is most frequent between twenty and forty.

*Symptoms*.—The majority of cases are of the nature of a chronic bronchitis, with special features, but, especially in children, acute attacks lasting from one to four weeks occur. Usually the onset resembles simple bronchitis, with a dry cough or with slight mucous expectoration, and slight constitutional disturbance, after a variable period, with the formation of the fibrinous casts, severe hacking cough, dyspnoea, rapid breathing, and pyrexia supervene, followed by the expectoration of the arborescent moulds.

The sense of suffocation and lividity may be pronounced if the bronchi are extensively im-

phreated, and the patient frequently complains of pain in the side, but with the expectoration of the casts there is generally an immediate temporary relief. Expulsion of the casts is followed by hæmorrhage from the bronchi, varying in amount from a few streaks to several ounces. The casts appear in the sputum rolled into a solid mass, mixed with ordinary mucopulverulent matter, but when placed in water the casts unroll and display their characteristic form.

In severe cases the interference with respiration is so considerable that total suffocation may ensue before the casts have been expelled. In other cases the course of the affection is less severe, and the attacks may last for weeks or months, or recur at intervals for many years.

The illustration on p. 14 is from a specimen of those finely branched fibrous casts in the Museum of the Royal College of Surgeons, London. They were expectorated by a boy aged eleven. He had always been delicate, and when about six years old had an attack of influenza. From that time he was subject to cough and expectoration, and spat up pieces of membrane at intervals. His mother's family was healthy, but on the father's side there was a strong tubercular tendency, and two of the six children had died, one of croup, one of consumption.

The physical signs are those of severe bronchitis, together with those resulting from more or less extensive occlusion of the bronchi. The casts never extend to the trachea, and rarely exceed a goose-quill in size. If only a few smaller bronchi are involved, special physical signs may be imperceptible, but usually there is absence of breath-sounds over the implicated areas, firstly from the blocking of the bronchi, and secondly from collapse of the corresponding air-vesicles. If the area involved is extensive, there may be dullness on percussion here, either hyper-resonance elsewhere, or the respiratory movements may be diminished, and retraction of the lower chest-wall may be present during inspiration. As the casts become loosened, râles or sibulant or whistling bronchi may appear, and a flapping sound has been observed.

The casts vary in length, from being mere fragments, to as much as four or five inches, or even, as in a case of Riegel's, six inches, but generally they are one or two inches long, and, when washed free from adherent mucus and suspended in water, form a perfect reproduction cast of the part of the bronchial tree. The larger stem is less in circumference than the tube in which it is formed, it rarely exceeds a goose-quill in size, and following the subdivisions of the bronchi, extends downwards to their finest ramifications, so that, according to Bietmer, the minutest terminations may be bulbous from being moulded in the infundibula. Excepting the smaller filaments, the casts are hollow, the lumen being usually filled with

mucus and bubbles of air. A transverse section shows that the casts are evidently deposited in successive layers, for they are always found to consist of concentric laminae of a fibrillated or hyaline basis, with numerous epithelial cells, leucocytes, oil-globules, granular débris, occasional Curschmann's spirals, and Charcot-Leyden's crystals in its meshes; blood-cells may be found on the surface. The disease is commonly termed fibrinous bronchitis, under the impression that the casts are composed of fibrin, but it has been shown by Grandy that they are composed of mucus, and not of fibrin, and are analogous to the casts of mucus colitis. Wilson Fox states that they are soluble in alkalis and in lime-water.

**Treatment.**—Little can be said in favour of any special drug or method of treatment in these cases, but during the attack the patient should be placed under the general conditions and ordinary treatment of acute bronchitis. Inhalations of ammonia, of finely sprayed lime water, of solutions of an alkaline carbonate, and the internal administration of iodide of potassium, creosote, turpentine, are said to have been employed with success.

Emetics may favour the expulsion of the casts, and Osler suggests pilocarpine might be useful, as it increases the bronchial secretion.

In the intervals between the attacks general hygienic measures and the use of tonics may perhaps prevent the attacks being so trying to the patient, but Walshe believes that no drugs, nor the best of health, nor the most favourable climates have any beneficial influence in modifying or preventing the attacks.

The varying phases of chronic bronchitis render it difficult to lay down any definite course of treatment, and the remedies and method of treatment of chronic bronchitis are so very numerous that it is only possible to mention a few, while endeavouring to suggest the lines of treatment that may be most suitable for the great variety of cases in which chronic bronchitis is the main feature. The most essential point is to discover, and, as far as practicable, remove, the cause of the bronchitis in any particular case. Thus if the affection is due to the inhalation of irritating particles of dust in his work, it may be necessary for the patient to change his occupation, if it is due to damp and unhealthy surroundings, he must be placed in a more suitable environment, if to Bright's disease, renal inadequacy, valvular heart-affection, syphilis, or gonit, treatment appropriate to these diseases is essential for relief.

In the milder and more chronic cases reliance should be placed mainly on general hygienic treatment, avoiding when practicable the use of drugs, unless specially indicated or required for the purpose of overcoming any complication or constitutional taint. But acute or subacute exacerbations generally call for more active

treatment on lines similar to that in acute bronchitis.

We may briefly consider the methods of treatment under four headings (1) General and climatic, (2) medicinal, (3) local applications, (4) counter-irritation, massage, baths, etc.

(1) *General and Climatic.*—The need for warm clothing, with flannel garments next the skin suited to the season, seemingly so obvious, is not always observed, and may have to be impressed on the patient, while the greatest care should be taken to avoid exposure to cold winds and rapid changes of temperature. Abundance of fresh air, either out of doors, or, if that is impossible, in warmed and well-ventilated rooms, is of the first importance. Too often sufferers from bronchitis increase their susceptibility, and even directly maintain their complaint, by shutting themselves up in close, stuffy rooms. Apart from the various affections which may be the essential cause of the complaint, any impairment of the general health tends to prevent recovery, thus any co-existing affection demands attention, especially does this apply to dyspepsia and constipation.

Nothing more generally proves beneficial than change of climate. Many cases do well by the sea-side. In England the south coast furnishes many suitable places in the winter, such as Torquay, Falmouth, Bournemouth, Hastings, the Scilly Isles, Isle of Wight, while the French Riviera, Biarritz, Madeira, or the northern African shore may prove more suitable to those who are able to go farther afield. But it is well to remember that a very large number of persons are always prone to derangement of the liver and stomach at the sea-side, and that this is one of the conditions which it is very necessary to avoid in bronchitis. For these a warm, equable climate such as that of Malvern, Chilton, or dry hill-air, or the Lake of Geneva, Bordighera, or Egypt may be better suited. Various Continental spas, such as Ems, Soden, Mont Dore, Carlsbad, Spa, etc., are beneficial, especially in gouty cases.

The diet should be light and nutritious, and the state of the stomach and liver, and any tendency to constipation, should receive careful attention.

(2) *Medicinal.*—In the acute exacerbations and in the very chronic cases the secretions tend to be scanty and the expectoration very tenacious. In these conditions, for increasing the expectoration and making it more fluid, iodide of potassium, carbonate of ammonia, apomorphia, iperacuanha, cocillina, and citrate of potash are useful, e.g. R Ext cocillina ℥ ℞x, apomorph hydrochl gr.  $\frac{1}{10}$ , syr prun virg ℥j, aq dest ad ℥ss. Tert quæ hor. Or, Ammon carb grs iv, tinct scyllæ ℥j, aq. chlorof ad ℥ss. Quatt quæ hor.

If there is a tendency to bronchial spasm, iodide of potash, caffeine, lobelia, myrtus eucan,

grindelia robusta, or bromide of ammonium may be especially indicated. When the cough is excessive and aimless, and due largely to mere bronchial irritation rather than the amount of secretion to be expectorated, some sedative should be combined with the other remedies, such as morphine, codeine, compound tincture of camphor, belladonna, stramonium, hydrocyanic acid, etc.—*e.g.* R Ext liq grind rob, ext liq myrtus chekan, aa ℥ss-xx, tinct lob eth ℥ss, aq chlorof ad ʒj. Quatt quæ hora.

When, on the contrary, the bronchitis is associated with profuse mucopurulent expectoration, the various preparations of opium and other direct sedatives should never be given without the most careful consideration, and then only with great caution, and in combination with some stimulant expectorant, such as the carbonate or chloride of ammonium with squill and senega. In these cases there is often considerable general weakness, for which iron, quinine, arsenic, and dilute mineral acids are desirable, especially in those cases in which the expectoration continues to be excessive despite the exhibition of the foregoing remedies, and in these conditions resort may be had to various gum resins and other expectorants, examples of which are the balsams of Peru and tolu, Canada balsam, ammoniacum, copaiba, cubebæ, creasote, tur or tur-water, terebene, turpentine, terpine hydrate, oil of sandalwood, etc., etc. Wilson Fox has recommended the tincture of lary Europææ as a valuable remedy in this class of cases, and also sulphur given internally as an electrolytic, combined with bitartrate of potash. For the chronic bronchitis of the aged, Whitla has found ammoniacum a most valuable expectorant, it relieves wheezing and promotes expectoration.

Cold-liver oil is often singularly valuable in very chronic cases, especially in those attended with profuse expectoration. Not only does it improve nutrition, but its use is often followed by a remarkable diminution in the secretions of the bronchial mucous membrane when all other remedies have been tried without effect. Nor should we forget that the weakness and dilatation of the right side of the heart, with which chronic bronchitis is so often associated, calls for such remedies as digitalis, strophanthus, and other.

(3) *Local Applications*.—Local applications include various inhalations and sprays, and intratracheal injections. For steam inhalations, benzoin, terebene, oil of Scotch pine, and creasote are commonly employed, thus a fluid drachm of the compound tincture of benzoin may be mixed with a pint of hot water at 104° F and the steam inhaled. If there is pain or bronchial spasm, two or three minims of chloroform, camom, or laudanum may be added. With the pine oils and creasote it is well to add carbonate

of magnesia, a useful formula being creasote, ℥ss, or oil of Scotch or Swiss pine, ℥ss-lx, light carbonate of magnesia, 20 to 30 grs, water to 1 fl oz for each inhalation. Atomised aqueous solutions of ipecacuanha wine as recommended by Murrell, chloride of ammonium, very weak tur-water, used with a Richardson's or other form of atomiser, or terebene, eucalyptol, menthol, or thymol, dissolved in vaseline oil, may be mentioned, as well as the wearing of a respirator with a small quantity of cotton-wool or sponge upon which has been dropped some of the volatile expectorant.

The value of intratracheal injections, by means of a syringe with a long suitably curved vulcanite nozzle, in certain cases merits careful consideration, the method is little practised, but is highly commended by those who have had recourse to it. It is particularly in patients with tenacious mucopurulent expectoration, and in putrid bronchitis and bronchiectasis, that the method is called for. Guided by a laryngoscopic mirror, the nozzle is gently but quickly passed below the vocal cords and the syringe emptied. Commencing with a drachm for each injection, the patient soon learns to tolerate two or three drachms being thrown in at a time. An oily menstruum, either olive or almond oil, or liquid vaseline, in which menthol, terebene, eucalyptol, or creasote have been dissolved (about three to five grains or minims to the fluid drachm, separately or in combination), forms a suitable injection. (Gianger Stewart's formula for bronchiectasis is menthol, ten parts, guaiacol, two parts, with olive oil, eighty-eight parts, a fluid drachm being injected daily. Another suitable formula is—chloriotorm, ℥ssss, balsam of Peru, ʒj, oil of eucalyptus, ʒj, castor oil to ʒj, half a drachm being injected once or twice daily.)

(4) *External Applications, Massage, and Baths*.—One of the most important means of relieving chronic bronchitis is counter-irritation, long continued rather than severe. Of various stimulating liniments, we may mention the compound camphor liniment, turpentine liniment with acetic acid, or a liniment containing cantharides or capsicum, or the application of iodine in solution, or mustard oil will be suitable for the purpose. The vigorous rubbing of the skin in the application of liniments is no doubt in itself beneficial. Nor should we overlook the undoubted value of systematic massage and Swedish exercises in a large proportion of cases. In young adults, cold baths followed by friction with a coarse towel, needle-baths, in fact a course of "hydropathic treatment," may be helpful in restoring vascular tone and the patient's general health, while materially assisting in the treatment of the bronchial affection.

### III BRONCHITIS IN CHILDREN

*ETIOLOGY*.—Bronchitis occurring in children,



while essentially similar to the affection in the adult, is nevertheless distinguished by certain predominating clinical features. The actual cause of acute bronchitis in children is usually exposure to cold, but it is in the poorly clad, under-fed, ill-housed children of the poor who are brought up in dirty, ill-ventilated, overcrowded rooms that the worst forms are observed. Individual predisposition largely influences the nature of a catarrhal attack, and while one child is prone to catarrhal affection of the upper respiratory tract, another tends to suffer from bronchial affections, and, again, others from gastro-intestinal catarrh.

Rickets, dentition, and intestinal catarrh are important etiological factors, and, as Ashby remarks, during the time that a tooth is being cut, children seem very apt to suffer from catarrh, which in winter affects the bronchial tubes, and in summer the intestines. Pressure of the tooth on the gums seems to act reflexly in producing a catarrh, sometimes with more or less spasm as the child becomes wheezy at night, sibilus being heard all over the chest, while in the morning it will be perfectly well.

A not unimportant factor in the occurrence of bronchitis is buccal respiration from nasal stenosis. The susceptible bronchial mucous membrane of children is unable to withstand the habitual inspiration of air unwarmed and unmoistened by normal nasal respiration, and consequently frequently recurring bronchial attacks are generally observed in children with post-nasal growths.

Whooping-cough and measles are generally accompanied by bronchitis, the bronchitis very often attacking the finer tubes, and being mainly responsible for the mortality of these diseases in young children.

**SYMPTOMS.**—Mild, uncomplicated bronchitis in children is attended by much the same train of symptoms as in the adult, but there is a much greater tendency for the catarrh to involve the finer bronchial tubes and the air-vesicles, a condition which is always attended with grave symptoms and considerable risk to life, owing to the weakness of the muscles of respiration (including the bronchial muscle) and the yielding nature of the chest-walls, features which more especially characterize rickets. In slighter attacks the pulse is moderately quickened, and the temperature raised two or three degrees above normal, but whenever the bronchial tubes are extensively implicated, the pulse is hard, the temperature is four or five degrees above normal, the respirations are quick, sometimes amounting to 70 or 80 a minute, with the alae nasi distended and working. The chest is expanded in the position of inspiration, the shoulders raised, respiration being chiefly abdominal, and the accessory muscles are brought into play, and the air failing to distend the lungs, the dyspnoea is attended with recession of the chest-walls,

especially of the epigastric and lower lateral regions. The skin becomes hot and dry, and the child restless and tossing, first to one side and then to the other, or having to be carried about, and constantly changing its position.

The invasion may be ushered in by rigors or convulsions, and convulsions not infrequently occur in the last stages of fatal cases.

It is only the milder cases which escape complications, especially collapse of the lung, broncho-pneumonia, bronchocystis, and emphysema, but in young children the onset of the attack may be severe, with great prostration, rigors or convulsions, bronchial spasm, pallor and lividity, and pronounced dyspnoea, which may rapidly asphyxiate the child before there has been time for the development of complications.

The pathological processes which result in collapse of the lung have already been described above (see p. 15). The symptoms are not very distinctive, unless the collapsed areas are in the aggregate sufficiently extensive to produce obviously increased respiratory embarrassment, falling in of the chest-wall, and dullness on percussion, and even then it is always difficult, if not impossible, to eliminate the coexistence of broncho-pneumonia, which in such cases is itself the chief cause of the severer symptoms.

The occurrence of broncho-pneumonia in the course of acute bronchitis may be suspected from the exaggeration of all the symptoms which usually result. "The child is restless, the cough shorter and more hacking, the skin hot and dry, the evening temperature usually reaching 103° or 104° F with morning remissions of several degrees, so that the fever assumes a remittent type, the dyspnoea is usually great, the respirations numbering forty or fifty, but varying with the amount of fever and extent of the lung involved" (Ashby). Examination of the chest unfortunately is often of very little assistance in the diagnosis of this complication, for in place of dullness on percussion there is more usually hyper-resonance owing to the emphysematous lung surrounding the pneumonic patch, and it is rare to find complete dullness, indeed, a considerable amount of pneumonia may exist in patches without obvious diminution of the percussion resonance. Ashby draws attention to the fact that while rhonchi are heard all over the chest, over the pneumonic portions rales of a consonant or ringing character may be heard, which are more intense and ringing because they travel to the ear through consolidated lung, and he states that even though no consolidated lung can be detected by percussion, the presence of consonant, intensely ringing rales with a temperature of 103° or 104° points almost certainly to pneumonia. Walsh points to the greater diffusion of the moist rhonchi in bronchitis as compared with pneumonia.

**THE DIAGNOSIS** of acute bronchitis in children is, as a rule, easy if suspected and sought for,

although there is seldom any expectation to be seen, inasmuch as young children almost always swallow what they cough up. There may be considerable laryngitis and glottic as well as bronchial spasm, sometimes causing confusion with spasmodic croup, but in bronchitis the spasm is not so marked, and the cough is less brassy and ringing in character. Laryngismus stridulus is a nervous affection, which, like bronchitis, is especially prone to occur in the subjects of rickets, but it is accompanied by other characteristic signs, and is, moreover, unattended with pyrexia, which is never absent in acute bronchitis in children.

Simple bronchitis in children, as in adults, may also be simulated by the invasion of measles, whooping-cough, typhoid fever, generalised tuberculosis of the lung, and more rarely by scarlatina and small-pox. The chief points of distinction have already been mentioned in the section on acute bronchitis in adults, but it is especially in children that difficulty in the earlier recognition of measles and whooping-cough is likely to arise.

**Prognosis.**—Acute bronchitis is especially fatal during the first two years of life, and, speaking generally, the younger the patient the more likely is a fatal termination, particularly in rickety children. The chief source of danger is the liability to involvement of the finer bronchial tubes, with asphyxia from extensive pulmonary collapse and broncho-pneumonia. Yet children will sometimes recover from a condition that is apparently hopeless. Hilton Fagge states that we must be cautious in giving an unfavourable prognosis in children; it is surprising how rapid may be both the pulse and the breathing, for two or three days together, in those who ultimately recover completely.

**Chronic bronchitis** in children is comparatively rare, since the processes of repair are more active in them than in adult life, while the very acute and severe attacks of acute bronchitis, from which only incomplete recovery is generally possible, frequently end fatally before the chronic stage is reached. Nevertheless, susceptible and delicate children are very prone to suffer from frequently recurring attacks of acute or sub-acute bronchitis, which ultimately pass into a more or less persistent chronic affection. In the milder cases they often lose their tendency to bronchitic affection about the age of puberty, and ultimately become strong and robust.

When children are the subject of persistent chronic bronchitis, it is almost invariably accompanied by emphysema and bronchiectasis. The chest becomes barrel-shaped, the shoulders rounded and high, the face pinched, and the body thin. The heart is dilated, the jugular veins large and prominent, and more or less cyanosis is usually observed. They are very liable to be carried off by an intercurrent acute attack, and are apt to become tuberculous.

**Treatment.**—The treatment of bronchitis in children is the same in principle as in the case of adults. The temperature of the apartment to which they are confined should range from 64° F. to 66° F., and in all but the milder cases the air should be kept moist by means of a steam-kettle. A linseed and mustard poultice, or hot fomentations, should be applied at the outset. It is necessary to be careful not to apply poultices or fomentations too hot for the tender skin of children, or to use much mustard in the poultice, and in infants and young children it is well to confine poulticing to the back if there is great difficulty in breathing, as the weight of a poultice may add a fresh danger instead of producing the relief desired.

The drugs most relied on are *pecuarina* in the form of the powder or wine, syrup of squills, *apomorphia*, and *antimony*. Small frequently repeated doses of the tincture of antimony or of *apomorphia* will generally relieve congestion and aid secretion in the earlier stages. It is undesirable to push these or any drugs to the point of vomiting or even nausea in the early stages. Small doses of *aconite* or *belladonna*, a minim or half-minim every two hours, are also of great service at this early period. *Belladonna*, recommended by Ringer and Murrell, has the effect of diminishing the secretion in the bronchial tubes, and on this account is often of much value in the bronchitis of children, when from the free secretion both into the bronchi and the pulmonary tissues there is a risk of broncho-pneumonia. It is much praised on this account by Coats in the treatment of broncho-pneumonia, and I am able to confirm his good opinion of it from my own experience. The drug should be given with my timid hand, the extract in doses of  $\frac{1}{4}$  to  $\frac{1}{2}$  being preferable to the tincture, but its effect must be watched, as it sometimes fails to relieve the respiratory embarrassment. If cough is excessive, small doses of bromide of potassium may be given. If opium or any of its preparations are considered necessary, they must be used with great caution. Non-depressing emetics such as *pecuarina* or sulphate of zinc are sometimes called for in the initial stages before the secretion has occurred, when the breathing is much impeded from the swelling and engorgement of the mucosa; they then may relieve the condition by aiding free secretion and by emptying an overloaded and distended stomach. Again, in later stages, when the tubes are becoming choked by excessive secretion which the little patient, either from weakness or from the tenacious character of the mucus, is unable to cough up, emetics are of signal service.

The diet should be light and nourishing, such as beef-tea, milk and milk-puddings, Benger's food, and barley-water, and in all cases the condition of the stomach and bowels should receive attention.

The importance of warm clothing, fresh air, and protection from cold and damp as prophylactic measures is obvious, and only requires mention

#### INSANITY OF CYANOSIS

A brain supplied with only partially aerated blood, like other organs of the body under similar conditions, is incapable of perfect functional activity, and the greater the coexistent defects in the brain structure the more pronounced is the functional aberration. Thus it is easy to understand that it is especially in persons of advanced years that mental obscurity, confusion and delirium, and various intellectual and emotional phenomena are apt to arise from prolonged or acute cyanosis in bronchitis and other pulmonary affections, and in various forms of cardiac disease. Clouston states that "the insanity of cyanosis from bronchitis, cardiac disease, and asthma is a form of delirium with confusion, hallucinations of sight, sleeplessness, sometimes suicidal impulses and vague fears. These symptoms are usually worse at night, and often end in mental torpor, passing into coma, and in some degree the mental power is usually affected in most old persons with diseases that prevent the blood being properly oxygenated."

But there are minor degrees of mental disturbances also due to cyanosis which do not amount to actual insanity, evidenced by change of manner, motivity, suspicion. Maudsley, in discussing mental changes due to impurities in the blood, distinguishes three changes—firstly, a general disturbance of physical tone; secondly, the engendering of chronic delusions; and, thirdly, in the more acute forms, acute delirium. In the aged the cyanosis and dyspnoea of capillary bronchitis cause muttering delirium or delusions, but in young, strong adults acute maniacal excitement is sometimes observed.

From the insanity of cyanosis we must distinguish those curious cases in which "alternation" between insanity and, for instance, bronchitis or asthma takes place. "There are certain cases of chronic bronchitis so called which, by their periodicity and by their chronicity, appear to have a distinct relationship to nervous disorder, and, in confirmation of this, we would say that just as certain cases of asthma alternate with other neuroses, so these cases of chronic bronchitis alternate with the neuroses. Asthma may be distinctly found to alternate with insanity, as a rule the insanity with which it alternates is of the melancholic type" (Hack Tuke). The same alternation is sometimes observed in chronic bronchitis of the type just mentioned, and Tuke refers to one such case in whom there had been two attacks of melancholia, and each of these was associated with freedom from the recurrent bronchitis.

#### Bronchiectasis

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See also BRONCH, COUGH, EXPECTORATION, HÆMOPTYSIS, LUNGS, GANGRENE OF, LUNGS, SYMPHYSIS OF, OSTEO-ARTHRITIS (Pulmonary), PLEURA, DISEASES OF (Empyema)

**DEFINITION.** Bronchiectasis or dilatation of the bronchial tubes is secondary to, or occurs as an incident in, a variety of pulmonary diseases. In some cases the condition is masked by the primary disease and unrecognised during life. This is especially the case in the acute forms when the smaller bronchioles are dilated (bronchiolectasis), and in the chronic form of the disease where there is general and fusiform dilatation of many tubes.

In the more typical cases the disease is easily recognised by certain prominent and characteristic features which entirely obscure the primary disease.

**ETIOLOGY AND GENERAL CAUSATION.**—In the histories of patients who are the subjects of bronchiectasis we find the following diseases—

1 Acute or chronic inflammatory diseases of bronchi, lungs, or pleura

(a) Acute and chronic bronchitis with or without emphysema

(b) Broncho-pneumonia, acute and chronic pleuro-pneumonia

(c) Chronic pleurisy and empyema

2 Obstruction, compression or stenosis of bronchial tubes

(a) Obstruction by foreign bodies in the bronchi

(b) Compression by aneurysm, hydatid, malignant, glandular, or congenital tumours (e.g. dermoid cysts)

(c) Stenosis as a result of syphilitic or other ulceration, with consequent stricture

3 Chronic lung diseases (tubercle, cancer, etc.) producing secondary bronchiectases, partly by inflammatory process, partly by compression

In some cases, however, no previous history of disease can be traced, and it has been suggested that some congenital weakness of the bronchial wall or deficient elasticity of lung has existed.

Much discussion has taken place upon the mechanical cause of dilatation of bronchi, and without entering fully into the many theories propounded, it may be taken for certain that two general conditions are usually present—

1 Some weakening of the bronchial wall, either congenital or more commonly of inflammatory origin

2 Some alteration in the normal conditions of pressure either within or without the tube

**INDUCING CAUSES**—There is no doubt the forced *expiratory efforts* associated with coughing are an important factor in bringing about the yielding of an already weakened wall, though it is impossible to eliminate the effects of dyspnoea with increased *inspiratory effort*. The accumulation of secretion combined with the effects of gravitation doubtless have their due share in some cases

**Morbid Anatomy and Pathology**—The leading features will be readily appreciated by reference to the points indicated under *Etiology*. Thus the appearances vary with the cause, the duration, and the presence of complications (pyæmia, hæmorrhage, etc.). Two general forms are recognised: (1) the *cylindrical or fusiform*, (2) the *globular or sacculated form*. The bronchial walls may be attenuated and atrophied, and the surrounding lung tissue ratched or emphysematous. More commonly the walls are markedly thickened, the peribronchial tissue indurated, the surrounding connective tissue showing a true interstitial and reticular induritis, while the intervening alveoli contain catarrhal products, which later become organised. The chronic pneumonia thus induced is secondary to the affection of the bronchus. In other cases the lung consolidation is primary, and the bronchiectasis secondary. Thus a twofold induration of lung tissue occurs, sometimes confined to the neighbourhood of the dilated bronchus and presenting irregular patches of consolidation, at others spreading to the entire lobe. In acute bronchiectasis the whole lung may be riddled with cavities. This is well seen in children, when the lung is often sponge-like and honey-combed. In the fusiform variety long cavities are found, either radiating from the root of the lung to the periphery, or confined to one or both bases. In the sacculated form the cavity is usually basal, and it may be deep-seated or superficial, containing very offensive, purulent secretion. In rare cases the dilatation is apical, and is thus usually associated with tuberculous disease. Occasionally a bronchiectatic cavity may closely simulate a phthisical one, but the two can be differentiated by the presence of the basement membrane in the former. The other viscera—heart, liver, brain, kidneys, etc.—may show secondary changes which are, however, not characteristic of this condition. Thus we may find any or all of the pathological appearances of pyæmia, acute or chronic. Hæmorrhages of serous membranes, acute pleurisy or pericarditis, empyema, and metastatic abscesses of brain or other distant parts are not uncommon.

**CHARACTER OF THE SECRETION AND BRONCHIAL CONTENTS**—On standing the secretion forms two

layers, an upper frothy, yellowish or brown in colour, and a lower consisting of purulent and solid matter. The odour is extremely offensive, and must be smelt to be appreciated. Fæcal is the term that most nearly describes it, but a smell is difficult to define. The solid matter consists of pus-cells, granular matter, and micro-organisms. Crystals are sometimes found of leucin, tyrosin, cholesterin, and fatty acids with many oil-globules. Post-mortem, the dilated bronchi are often found to be filled with purulent, caseous looking material (see "Expectoration").

**CHRONIC BRONCHIECTASIS**—ACUTE BRONCHIECTASIS or bronchiectasis is usually a disease of children. Its symptoms cannot be distinguished during life from those of the capillary bronchitis with which it is associated.

CHRONIC BRONCHIECTASIS is a disease of children or of early or middle life, occurring more frequently in men than in women. When secondary to phthisis, cancer, or obstruction of bronchi from any cause, it may occur in either sex or at any age.

*Cylindrical bronchiectasis* is only to be differentiated from the chronic bronchitis or other disease of which it forms a part by the excessive bronchial secretion, or occasionally by fetor of the cough or expectoration (though this is by no means constant), and by the enlargement or clubbing of the terminal phalanges of fingers and toes, which is frequently but not invariably seen.

The characteristic symptoms of the disease are more commonly observed in the *sacculated bronchiectases*, less often in the *cylindrical*. Cough is a prominent symptom, and expectoration with excessive secretion of an extremely offensive and fætid odour.

The cough is frequently intermittent in character and occurs in violent paroxysms, somewhat resembling whooping-cough, and terminating in the expulsion of a large quantity of mucus or mucus pus presenting the characteristic features described. Sometimes one fit of coughing, or perhaps two, occur during the twenty-four hours, often induced by change of posture (such as rising from bed, stooping, lying down). It seems probable that the reason for the paroxysmal nature of the cough and large expulsion of bronchial secretion is due, in the few cases in which it is present, to the mechanical position of the cavity or cavities, which, when filled beyond a certain point, overflow on the least change of position into the neighbouring undilated tube, and set up an irritable spasm which leads to violent expiratory efforts and subsequent expulsion of the contents.

The sacculated tubes, being thus emptied entirely or partially, the patient may be free from cough for the rest of the day and comparatively comfortable. In other cases the cough does not exhibit this paroxysmal nature,

but is frequent, though still accompanied by excessive and often offensive expectoration. In children the attack is often mistaken for vomiting. In reality a slight cough is followed by retching and the sudden expulsion, not of vomit, but of foetid mucus, sometimes exceeding half a pint in amount. *Hæmoptysis* is not at all uncommon, but except in late stages of the disease is rarely excessive. The *temperature* of the patient is not raised unless the disease is associated with bronchitis or catarrh. A frequent though not invariable feature is the *clubbing* of the distal phalanges of fingers and toes. In rare cases other joints share in the enlargement (see "Osteo-Arthropathy, Pulmonary").

Nutrition is often well maintained, and the strength and appetite good. In later stages there may be wasting, excessive dyspnoea, anorexia, or alimentary troubles. None of the above symptoms are constant. There may be little or no cough or expectoration. Factor of expectoration, cough, or breath may be present or absent. Clubbing cannot be regarded as pathognomonic of the affection, though it is commonly present. In the later stages of the disease grave symptoms are often associated, due to catarrh or broncho pneumonia, or to ulceration of bronchial tubes. In the latter case acute septicæmia or one of the many manifestations of pyæmia may ensue. Death may occur from—

- (1) Acute bronchitis or broncho-pneumonia
- (2) Cardiac failure
- (3) Exhaustion often induced by vomiting or other alimentary affections
- (4) Amyloid disease and its accompaniments
- (5) Hæmorrhage from ulceration into or aneurysm of pulmonary vessels
- (6) Acute or chronic pyæmia with inflammatory or suppurative affections of serous membranes and metastatic abscesses, of which the most frequently recorded are abscesses of brain.

**PHYSICAL SIGNS.**—There are no pathognomonic signs by which we can distinguish dilated bronchi.

It will be remembered that in the acute cases in children and in some chronic cases of cylindrical bronchiectases, the signs are those of the associated bronchitis only. For the sake of simplicity the physical signs may be described under the following heads—

1 *Where the disease is masked by the deep position of the Dilated Bronchi or by Emphysema, and unaccompanied by surrounding Pneumonia.*—The chest may be resonant or hyper-resonant (in the case of emphysema). If the cavity approaches the pleural surface there is Skodac resonance or absolute dullness, according as the bronchial dilatation is filled with fluid secretion or not. In the former case breath-sounds at the base or bases (which are usually affected) are absent or feeble, and the voice-resonance diminished. When the dilatation is empty, the respiratory murmur becomes broncho-cavernous,

or cavernous with marked pectoriloquy. This variability in the physical signs is important and will be referred to later.

In the cases of deeply seated dilatations it is not uncommon to find that resonance, vocal fremitus, voice sounds, and respiratory murmur do not depart from the normal, or perhaps the breath-sounds are somewhat harsher than usual. Adventitious sounds may or may not be present. When present they vary from the small bronchitic crepitation to the large bubbling rale. In this and the next group of cases, however, a marked feature may be the extraordinary variety of size and sound of rale. When a cavity is superficial, bubbles and rales of every size, squeaking and "croaking" sounds, or merely silant or sonorous dry sounds may be present. The term "croak" applied by Ewart well defines the characteristic quality of the sound.

2 *Cases where the Dilatation of Bronchi is associated with a secondary Consolidation of Lung.*—Here the signs of consolidation, either patchy or confined to one or both bases, are superadded. Dullness to percussion, diminished or increased vocal vibration and resonance are present, varying with the condition of the pleura and with the depth of the dilated bronchus. When the case is well marked a feature is the variation in the degrees of increased voice-resonance as the stethoscope is passed from one spot to another at the base. In a tuberculous cavity near the surface there is frequently found a pectoriloquy of fairly uniform character and intensity over the whole surface of the cavity. It is not so where a series of bronchiectatic cavities occur at various distances from the pleural surface. Pectoriloquy of varying degree of pitch and loudness is heard at different spots on the chest-wall.

Further, as in the first class of cases, the physical signs vary from time to time with the full or empty state of the bronchial cavity, provided it is near the surface. Again, rales may be absent or numerous, and those that are considered by some authorities to be peculiarly characteristic of the disease are sometimes present, especially the "croak" previously referred to (described under different names by various authors), and a sound known as the "veiled puff" of Skoda, heard in rare cases at the end of inspiration. It cannot be positively affirmed that these two sounds are never heard in other pulmonary diseases, but it is certain that they are more often associated with bronchiectasis.

3 *Cases in which the Consolidation or other Disease of Lung is primary, and the Dilatation of Bronchi a secondary result.*—Space will not allow a detailed description, which would indeed be an account of the physical signs of consolidation or fibrosis of lung due to an old pleurisy or empyema, a chronic pneumonia, tuberculous fibrosis, or any indurative pulmonary disease,

or, again, of the signs of obstruction of bronchus or of some tumours, malignant or otherwise, producing compression.

Retraction of the side and displacement of the heart are usually the result of a contracting fibrosis or of an old pleurisy, and are not found so frequently in the second group, where the organising pneumonia is secondary to the dilatation of bronchi. When the disease is confined to one or both apices of the lungs the case is usually a tuberculous one, though this is not an invariable rule.

**Diagnosis.**—(a) *From Tuberculous Disease of Lungs.*—In the early stages the comparative well-being of the patient, the absence of wasting and of pyrexia, and the common association of clubbing of fingers (more rare in tuberclosis) serve to distinguish the case from one of tuberculous disease. In phthisis a basic disease, moreover, is of extreme rarity, unless the apex has been primarily affected. Tuberculous cases, however, do occur with fibroid consolidation of one lobe. These are usually of pleuritic origin, or are found in rare cases of simple tuberclosis.

The absence of tubercle bacilli is to be assumed. It must not be forgotten, however, that bronchial dilatation with its characteristic symptoms is occasionally a companion of tuberculous disease, especially in those forms where there is much induration. The diagnosis is more difficult when there is bronchiectasis with chronic pneumonia of the upper lobe, especially if haemoptysis be also present. In such cases the physical sign (upon which stress has been laid) of the variability of voice-resonance, and even of respiratory murmur in different situations at the apex, becomes of importance. The changes due to the emptying and refilling of a cavity are sometimes found in a basic tuberculous excavation, but far more often in bronchiectasis. These signs are less likely to occur at the apex of the lung.

(b) *The discharge of a localised or small basic empyema into a bronchus with the formation of a permanent sinus is to be distinguished from bronchiectasis by the history of the disease, and usually by the absence of factor of the mucopurulent or purulent discharge.* The diagnosis is not always easy, because in an old discharging empyema the fingers and toes are often extremely clubbed, the discharge is intermittent and occasionally blood-stained and fetid. The factor of expectoration is usually a sign that more than a mere sinus is present, and that probably as a result of the pleurisy and consequent emphysema of lung some of the neighbouring bronchi have become dilated.

(c) *Chronic Bronchitis.*—As we have previously stated, it is often impossible to diagnose dilated bronchi in a case of chronic bronchitis, though it may be conjectured that the condition is present if the sputum becomes fetid and the fingers clubbed. Most authors describe a form

of bronchitis with offensive sputum under the name of fetid bronchitis or bronchorrhœa. The writer is of opinion that this disease is a myth, and that factor of the bronchitic sputum in cases of chronic bronchitis means nothing more than that some of the bronchi are the subjects of fusiform dilatation, and that owing to the accumulation of secretion and consequent decomposition the sputum becomes offensive. When the fingers are not clubbed, care must be taken not to confound factor of breath, cough, or sputum with that due to other causes. Pharyngeal mucus is sometimes excessive and of extremely offensive odour, but the discharge is usually in the early morning. Oræna and the fetor from carious teeth and the offensive breath from constipation with disturbed liver must all be eliminated.

(d) *Gangrene and Abscess of Lung.*—*Gangrene* is an acute and grave condition, and could only be confounded with the later and ulcerative stages of bronchial dilatation. As a rule, the history aids the diagnosis. The gangrenous odour is usually distinct and characteristic, but may be confused with that of bronchiectasis. Some aid is also to be obtained from an examination of the sputum. In gangrene there is more destruction of tissue, and elastic fibres are frequently found, whereas the expectoration from a bronchiectatic cavity rarely contains them. If gangrene is associated with tuberclosis, tubercle bacilli will also be present. As in the case of gangrene, the formation of a *pulmonary abscess* is sudden in its onset. Whether a sequela of pneumonia or a result of pulmonary embolus, the disease is acute, and usually an evacuation of pus is followed by a quiescence of symptoms.

Cases, however, occur of a basic pulmonary abscess in which factor of expectoration, intermittent cough, and discharge of pus and clubbing of fingers are present. They are extremely difficult to diagnose from a sacculated bronchiectasis. The factor is not so pronounced as in gangrene. Several cases have been recorded in which operative procedure has resulted in complete cure. A small localised empyema discharging through the lung, or a sub-pleural chronic abscess, may easily be confounded with a single sacculated dilatation of a bronchial tube.

**Prognosis.**—Bronchiectasis is usually an extremely chronic disease. In children the acute cases associated with capillary bronchitis rarely recover. The condition of the heart and circulation (especially right heart) is an important element in prognosis. The onset of pyrexia, acute or chronic, profuse hæmoptysis, or cerebral symptoms are of grave significance, and usually precede a fatal termination. In addition to the many complications of the disease already referred to, death may result from exhaustion.

**Treatment.**—*General Considerations.*—Attention must be paid to the nutrition and strength

of the patient. A dry, warm, or equable climate, and remedies that improve the appetite and nutrition, are advisable. Amongst such remedies may be included cod-liver oil and malt extracts, the hypophosphites and glycerophosphates, with or without one of the following tonics: quinine, iron, or strychnine. Two drings appear to act in a twofold manner. Benzol in five- to ten-minim doses in a flavoured emulsion, and creasote in capsules of one, two, or more minims, not only increase appetite and improve the digestion, but probably act as antiseptics. They are both easily tolerated after the first few doses. Any or all of these, especially if combined with the influence of a dry, pure air, increase the vigour and recuperative power of the patient, and probably lessen the tendency to catarrh.

*Complications.* *Bronchitis and catarrhal affections* of the lungs must be treated on general principles. *Cardiac failure* will need the administration of digitalis or cardiac tonics. Care should be taken in the case of systemic engorgement to encourage the action of the liver secretion by administration of small doses of mercury or cholagogue purgatives, and to promote diuresis. *Alimentary troubles* are to be dealt with by sedative treatment or digestive tonics, with bland, unstimulating nourishment. If *hæmoptysis* occurs, absolute rest in the recumbent position should be prescribed, unless the symptom is very slight. In case of profuse hæmorrhage, morphia may be given with caution, combined with remedies that diminish arterial tension, while the amount of fluid in the diet must be diminished.

*Special Treatment.* The object is to diminish bronchial irritation and inflammation and to promote antiseptics.

Guaiaol is of special value when given in doses of 3 to 5 grains several times a day in capsules or cachets. Terebene (5 grains in capsules), oil of turpentine (10 to 20 minims with milkage in an emulsion), benzoin (10 to 20 minims of the compound tincture), or benzoic acid (5 grains), ammoniacum or guaiacum (half to one ounce of the mistura ammoniaci or mistura guaiaci)—all these are found to be of service in diminishing chronic catarrh and in lessening the bronchorrhœa or excessive secretion.

Many of these remedies act directly through the blood, and some, like guaiaol, are probably eliminated through the respiratory mucous membrane. If large cavities are present in which secretion accumulates and decomposes, it is of the utmost importance that all the means at our disposal should be adopted to empty these cavities as frequently as possible. Without this the value of astringent treatment is diminished, and all the subsequent measures that are now to be described will be of little value.

*Methods of emptying Bronchiectatic Cavities.*—Change of posture will often produce violent

cough with the expulsion of a large amount of expectoration. In some cases the act of lying down, stooping, or even leaning out of bed is sufficient. The occasional paroxysmal attacks are rather to be encouraged than not.

*The Continuous Inhalation of Creasote.*—The continuous treatment by the inhalation of creasote has found great favour, and is probably more successful than any other method in bad cases. The patient should be placed in a special room or chamber, in which an ounce or two of ordinary commercial creasote is vaporised on a small metal bath heated by a spirit lamp. The patient's eyes are covered, and the nostrils plugged with cotton-wool. He inhales the dense fumes of creasote vapour for a quarter of an hour to twenty minutes, at first every other day. Ultimately he should be subjected to it for one hour to one hour and a half daily by slow movements in the length of treatment. Tolerance is soon established, and in many cases the relief is considerable. Violent cough and more or less complete emptying of the cavities is the result. Probably some absorption of the drug and possibly a certain amount of local effect are produced, though this is doubtful.

Occasionally the amelioration is rapid and decided, but in many cases the result of months of treatment is only a diminution of expectoration, and especially of the factor. Occasionally brilliant results have been produced. It may be asserted that better results have been obtained at the Brompton Hospital from the prolonged use of creasote inhalation than from any other form of treatment.

Provided the dilated bronchi can be partially or wholly emptied, other methods of treatment which are believed to have a local effect may be valuable. These are (1) *Moist Inhalations.* Creasote, turpentine, carbolic acid, or compound tincture of benzoin are mixed with hot water at a temperature of 140° in an inhaler. It is well known that though these remedies are deeply inhaled they do not reach far down the trachea. The plan of treatment is well recognised, but is not of permanent value.

(2) *Intra-laryngeal Injections.*—A 2 per cent solution of guaiaol or of creasote in menthol and olive oil injected daily or every second day is beneficial in some cases.

(3) *Treatment by Drugs which promote Antiseptics and diminish Factor.*—Guaiaol and creasote have already been mentioned. Garlic, 1 to 3 grains in cachet, or 1 to 4 drms of the syrup, either alone with equal quantity of syrup of tolu, or in combination with creasote and syrupus piceæ liquidæ, has also been recommended. The disagreeable odour following its use is a disadvantage.

*Operative procedures* have not been attended by good results on the whole. The opening and drainage of cavities at the base of the lung have been frequently tried, usually when it was

thought that there was a single cavity. The results are not often encouraging, because frequently multiple cavities have been found where one has been diagnosed. There is some danger of septic absorption into the surrounding tissues and the edges of the wound and sinus formed, owing to the putrid character of the evacuated secretion.

Indications for operation are found in the following signs:—(1) There must be evidence of a single cavity. (2) Given a single cavity, high fever and signs of ulceration of bronchi or of putrid absorption completely justify an operation, consisting of resection of portion of one or two ribs and the drainage of the cavity. Complete recovery has sometimes resulted.

**Bronchiectasis.** *Vide supra* BRONCHI, BRONCHIECTASIS.

**Bronchiocrosis.** *See* TABES DORSALIS (*Bronchial Crises*).

**Bronchiolectasis.** *See* BRONCHI, BRONCHIECTASIS.

**Bronchiolitis.** *See* LUNG, TUBERCULOSIS OF (*Pathological Anatomy*); TUBERCULOSIS (*Morbid Anatomy, Lungs*).

**Bronchospasm.** *See* ASTHMA (*Nature and Etiology*).

**Bronchitis.** *Vide supra*, BRONCHI, BRONCHITIS.

**Bronchitis, Capillary.** *See* PNEUMONIA, CLINICAL (*Complications*).

**Bronchocele.** *See* THYROID GLAND, MEDICAL (*Goutie*).

**Broncholith.** A concretion in calcareous formation in a bronchial gland or tube.

**Bronchophony.**—The sound of the voice heard by means of the stethoscope placed over the course of the trachea and bronchi, also the same sound heard, as a pathological condition, over lung tissue in a morbid (consolidated) state. *See* CHEST, CLINICAL INVESTIGATION OF (*Auscultation*); PNEUMONIA, CLINICAL (*Physical Signs*).

**Bronchophthisis.**—Pulmonary phthisis commencing in ulceration of the smaller bronchi.

**Bronchopleurisy.**—The association of bronchitis with pleurisy. *See* BRONCHI, BRONCHITIS, PLEURA, DISEASES OF.

**Bronchopneumonia.**—Inflammation of the lungs originating in the smaller bronchi, catarrhal pneumonia. *See* ALCOHOL (*Clinical Uses of*), BRONCHI, BRONCHITIS, BURNS AND SCALDS (*Respiratory Complications*), PNEUMONIA, BACTERIOLOGY, PNEUMONIA, CLINICAL.

**PULSE** (*in Pneumonia*), RICKETS (*Complications*), TYPHOID FEVER (*Complications and Sequelae*).

**Bronchorrhagia.**—Hæmorrhage from the bronchi. *See* HÆMOPTYSIS.

**Bronchorrhœa.**—Chronic bronchial catarrh with excessive secretion. *See* BRONCHI, BRONCHITIS (*Clinical Varieties*), BRONCHI, BRONCHIECTASIS, THYROID GLAND, MEDICAL (*Exophthalmic Goutie, Respiratory System*).

**Bronchoscopy.**—The use of the laryngoscope for the inspection of the interior of the bronchi, for the detection and removal of foreign bodies therefrom; it may be passed through the mouth and larynx or (better) through a tracheotomy wound, its use has been recommended and perfected by Kilham.

**Bronchotome.** An instrument employed in post-mortem examinations for laying open the bronchial tubes.

**Bronchotomy.**—An operation sometimes performed for the removal of foreign bodies from the large bronchi, in posterior bronchotomy, resection of some of the ribs between the scapula and the spinal column is carried out.

**Broncho-vesicular Breathing.**—The type of breath sound normally heard in the interscapular region and over the manubrium sterni, heard over other parts of the lung it may indicate early tubercular disease. *See* CHEST, CLINICAL INVESTIGATION OF.

**Bronzing of Skin.** *See* ADRENAL GLANDS, ADDISON'S DISEASE, DIABETES MELLITUS (*Symptoms, The Skin*), NEW-BORN INFANT (*Winkler's Disease, Bronzed Hamaturic Disease*), PEDIATRICS (*P. Copensis*), PREGNANCY, PHYSIOLOGY (*Local Changes, Antidromal Wall*), SKIN, PIGMENTARY AFFECTIONS OF (*Actinic Bronzing, etc.*), THYROID GLAND, MEDICAL (*Exophthalmic Goutie, Papulomatosis*).

**Broom Tops.**—Broom tops (*Scoparia Caerulina*) are used as a diuretic medicine, in association with other drugs, in dropsical conditions, but not in acute nephritis; they contain a diuretic principle (*Scoparin*), and an alkaloid (*Sparteine*), the official preparations are the *Infusum Scoparii* (dose, 1 to 2 fl oz) and the *Succus Scoparii* (dose, 1 to 2 fl dr).

**Brophy's Operation.**—A modification of the plastic operation for the cure of cleft palate and hare-lip introduced by Truman W. Brophy, it consists in early operation (within the first three months of life), in correcting first the cleft in the palate, and in separating the mucous membrane over each malar process (so as to permit approximation).

**Broth.** *See* BOUILLON, INVALID FEEDING.



(*Food in Convalescence*), POST-MORTEM METHODS (*Cultivation Media, Beef Broth*)

**Broussaisism.**—The doctrine of pathology and therapeutics introduced by François Joseph Victor Broussais (born 1772, died 1838), in it great stress is laid upon irritability, and especially that of the mucous membrane of the digestive tract. See BRUNONIAN SYSTEM.

**Brow Ague.**—Frontal (trigeminal) neuralgia, especially when malignant. See NERVES, NEURALGIA.

**Brow Cases.** See LABOUR, DIAGNOSIS AND MECHANISM (*Brow Presentations*), LABOUR, MANAGEMENT (*Brow*).

**Brown Atrophy.**—A degenerative change in the myocardium, met with in chronic valvular disease, etc. See HEART, MYOCARDIUM AND ENDOCARDIUM (*Morbid Processes, Atrophy*).

**Brown Induration.**—A state of passive, mechanical congestion of the lungs, met with commonly in heart disease, and also from the pressure of tumours, the lungs are large, "russet-brown" in colour, and show an increase in the connective tissue which they contain. See LUNGS, VASCULAR DISORDERS (*Passive Hyperæmia*).

**Brownism.** See BRUNONIAN SYSTEM.

**Brown-Séguard's Epilepsy.**—The convulsive phenomena which follow certain experimental lesions, such as hemisection of the spinal cord, division of the sciatic nerve, etc. See also EPILEPSY (*Etiology*).

**Brown-Séguard's Paralysis.**—The syndrome due to unilateral lesions of the spinal cord (e.g. in syphilis, tumours, hemorrhages, etc.), with paralysis on the same side as the lesion, and cutaneous anaesthesia on the opposite side. See SPINAL CORD, MEDICAL (*Brown-Séguard Anaesthesia*).

**Bruch, Membrane of.** See IRIS AND CILIARY BODY (*Anatomy*).

**Brucine.**—An alkaloid derived from the *Strychnos nux vomica*, acting as a local anæsthetic. See ALKALOIDS, NUX VOMICA.

**Bruiques.** See MEDICINE, FORENSIC (*Kinds of Wounds*), KNEE-JOINT, INJURIES OF (*Bruiques*).

**Brult.**—A name given to various sounds (murmurs) heard on auscultation over the heart, great vessels, lungs, œsophagus (sticture), and uterus (pregnant or with fibroid tumours). The *bruit d'aër* is the metallic echoing sound heard in pneumothorax, the *bruit de cuir neuf* is the "new leather" creaking murmur heard in fibrous pericarditis, the *bruit de sable* is the "humming-top" murmur or venous hum

heard in anemia and exophthalmic goitre, the *bruit de drapeau* is a dry râle due to the vibration of bronchial casts in fibrinous bronchitis, the *bruit de galop* is a sign of muscle failure of the heart, and consists of a triple rhythm of the cardiac sounds (double first sound and accentuated second), the *bruit de mouton* is a splashing, churning sound met with in pneumopericardium, the *bruit de pot fêlé*, or cracked-pot sound, is that heard, on percussion, over large thin-walled pulmonary cavities, and best with the mouth open, and the *bruit de souffle* is the common "bellows murmur" heard in endocarditis, etc.

**Brunner's Glands.**—Branched tubular glands of the duodenum discovered by Brunner (1653-1727), a Swiss anatomist. See INTESTINES, DISEASES OF (*Anatomical and Physiological Considerations*), PHYSIOLOGY, DIGESTION (*Structure of Alimentary Canal*), STOMACH AND DUODENUM, DISEASES OF (*Anatomy, etc.*).

**Brunonian System.**—A theory of medicine founded by Dr John Brown or Bruno, a Scots physician (1735-88), according to whom physical life and disease were due to a peculiar excitability (or irritability), excessive excitability produced sthenic diseases, and defective led to asthenic ones. It was opposed to the practice of blood-letting.

**Brussa.** See BALNEOLOGIA (*Turkey*).

**Brygmus.**—Chattering of the teeth.

**Bryocytic.**—A disease characterised by cell proliferation is said to be *bryocytic*, e.g. syphilis and cancer.

**Bryonia.**—The root of *Bryonia alba* and *B. dioica*, containing a glucoside or bitter principle (bryonin), it is official in the United States, it is used (rarely now) as a hydragogue cathartic and (externally) as a vesicant. The *Tinctura Bryoniæ* is given in doses of 2 to 10 fl dr.

**Buboes.** See VENEREAL DISEASE (*Soft Sores, Complications*). See also GROIN, PLAGUE, RHEUMATISM, SKIN DISEASES, SYPHILIS, etc.

**Bubonic Plague.** See PLAGUE.

**Bubonocoele.**—Any swelling in the inguinal region, but more especially a hernia. See HERNIA.

**Bucco.**—As a prefix "bucco-" signifies "relating to the mouth," and is used in such combinations as bucco-labial, bucco-pharyngeal, etc.

**Buchu Folia.**—The leaves of the *Barosma betulina*, the official preparations of which are the *Infusum Buchu* (dose, 1 to 2 fl oz) and the *Tinctura Buchu* (dose,  $\frac{1}{2}$  to 1 fl dr), it is used as a mild diuretic and vehicle for other

diuretics in catarrhal affections of the bladder and urinary tract, it gives a marked odour to the urine (Note Buchu is an indeclinable word)

**Buckthorn.** See CASCARA SAGRADA

**Bucnemia.** Elephant-leg (from *Ga* *Boñ*, augmentative particle, and *kripip*, the leg) See FILARIASIS (*Elephantiasis Arabum*)

**Budd's Cirrhosis.**—Dyspeptic (non-alcoholic) hepatic cirrhosis. See LIVER, DISEASE OF (*Portal Cirrhosis*)

**Bude.** See THERAPEUTICS, HEALTH RESORTS (*English*)

**Buffy Coat.**—In slow clotting of the blood (e.g. in fevers) the red cells have time to sink somewhat, leaving the upper part of the clot of a paler colour, the grey or "buffy coat" See BLOOD (*Coagulation*), PHYSIOLOGY, BLOOD (*General Characters*)

**Bug, Harvest.** See STINGING INSECTS

**Buhl's Disease.**—Acute fatty degeneration of the new-born infant, with hemorrhages in the heart, liver, and kidneys. See NEW-BORN INFANT (*Diseases, Buhl's Disease*)

**Buist's Method of Artificial Respiration.** See ASPHYXIA (*Resuscitation, Infants*)

**Bulam Fever.** See YELLOW FEVER

**Bulb.**—With the meaning of a somewhat spherical dilatation this term is widely used in anatomy and physiology, e.g. hair-bulb, bulb of the urethra, vaginal bulb, end-bulbs, bulb of the ovary, eye, etc. The bulb of the spinal cord is the medulla oblongata. Wet and dry bulb thermometers are used in hygrometry. See METEOROLOGY (*Hygrometry*)

**Bulbar Paralysis.**—A disease due to an affection of the neurones of the motor nerves of the medulla oblongata, glosso-labio-laryngeal paralysis. See PARALYSIS (*Bulbar Paralysis*), AUDITORY NERVE AND LABYRINTH (*Nerve Deafness*), MUSCLES, DISEASES OF (*Progressive Muscular Atrophy in Young Children*), SYRINGOMYELIA (*Symptoms, Diagnosis*)

**Bulgaria.** See BALNEOLOGY (*Turkey, Bulgaria*)

**Bulimia.**—Insordinate or voracious appetite, associated with faintness, bulimiasis. See APPETITE (*Increase*)

**Bulla.**—A bleb or blister. See SKIN, DISEASES OF, LEPROSY, PEMPHIGUS, etc

**Buller's Shield.** See CONJUNCTIVA, DISEASES (*Purulent Ophthalmia, Treatment*)

**Bullet Wounds.** See MEDICINE, FORENSIC (*Wounds from Firearms*), WOUNDS (*Varieties*)

**Bulpiis.**—A parasitic skin disease of the nature of ringworm, occurring in Nicaragua, and probably identical with the disease *Curete* of South America. See SKIN, PARASITES (*Trinea*)

**Bungarus.** See SNAKE BITES

**Bunge's Law.**—The fact that a parallelism exists between the ashes of the milk of the mother animal and the ashes of the foetus, it does not hold with regard to the human foetus and human milk

**Bunion.** See DEFORMITIES (*Hallux Valgus*)

**Buphthalmus.**—Literally "ox-eye", as a bulging or protrusion of the whole eye, with increase of tension, it is known also as megalophthalmus, congenital glaucoma, and hydrophthalmus congenitus, it is due to a malformation in the eye (e.g. want of separation of the iris from the cornea in the region of the ligamentum pectinatum [*vide* Ballentine's *Antenatal Pathology*, vol. II, 1904])

**Burdach's Column.** See SPINAL CORD (*Anatomy, Ascending Tracts, Dorsolateral Column*)

**Burdwan Fever.** See MALARIA

**Burgundy Pitch.**—Pix Burgundica, or the resin from the spruce fir, is used as a basis for plasters, it contains pinic acid, a volatile oil, etc., the official preparation is *Emplastrum Piceae*

**Burial-Places.** See also CREMATION—The disposal of the dead commonly takes the form of earth-to-earth burial, for cremation is not yet commonly practised and vault burial is much restricted. Burial-places or cemeteries should be situated in the suburbs of large towns, and should, at the same time, be easy of access. Old cemeteries in the centre of a town should be closed, they may be converted into open spaces, but must not be built upon (save for the purposes of enlarging a place of worship). A clay soil, a chalky one, and a loose gravel one are all unsuitable for a cemetery, the best kind of earth being a dry loam. The drainage of the surface soil should be thorough, there should be no wells in common use in the neighbourhood of the cemetery. Trees and shrubs should be planted. One-fourth to one-half acre per thousand persons of the population is the space which ought to be set aside in towns for the purposes of burial. Individual graves should not be more than eight feet deep, for dissolution of the body takes place more quickly near the surface of the ground, at the same time, four feet of earth should always cover an adult body, and

three feet that of a child under twelve years. Wicker-work or light wooden coffins are better than heavy wooden or leaden ones, for they permit a more rapid dissolution of the body. A foot of earth must separate two coffins in one grave, and no unvalled grave shall be reopened within fourteen years after the burial of a person above twelve years of age or within eight years after the burial of a child under twelve years of age, except to bury another member of the same family. Since it is undesirable and may be dangerous for dead bodies to remain awaiting burial in the living-rooms of the poor, it is desirable that all towns should be provided with *Mortuaries*.

The Acts of Parliament regarding burial-places (for England, Wales, and London) are the Burial Act (1853), the Public Health Act (1875), the Public Health (Interment) Act (1879), and the Disused Burial Grounds Act (1884). For Scotland there are the Burial Ground (Scotland) Act (1885), and the Public Health (Scotland) Act (1897).

If vaults are used at all they must be enclosed with walls of brick or stone solidly put together with good mortar or cement; they must not be disturbed. Vault burial, however, is not to be recommended. A grave which is walled with brick or stone work is regarded as a vault.

**Burking.** The murdering (usually by smothering) of individuals in order to sell their bodies for purposes of dissection. The name took its origin in connection with the notorious Burke and Hare case, or the West Port murderers of Edinburgh, in 1828.

**Burmese Ringworm.**—Tropical ringworm or Dhobie's itch. See SKIN DISEASES OF THE TROPICS (Caused by Vegetable Parasites).

**Burnett's Fluid.**—A solution of zinc chloride, having a sp gr of 2.0, used as a domestic antiseptic.

## Burns and Scalds.

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See also DEFORMITIES (*Club-Foot, Acquired*), EYEBALL, INJURIES OF (*By Heat*), GANGRENE (*Burns*), GROSS (*Injuries of, Burns*), MEDICINE, FORENSIC (*Burns*), RADIUM, SKIN GRAFTING, TEMPERATURE (*Depression*), X-RAYS.

The term *Burn* is applied to the injury resulting from the application of dry heat to the

tissues of the body, while the damage done by moist heat is usually spoken of as a *Scald*. The distinction is unimportant, as the essential characters of the lesions produced by the two forms of heat are for all practical purposes the same, and conditions almost identical frequently result from the application of such strong caustic substances as fuming nitric acid, caustic potash, nitrate of silver, or arsenical paste.

**CAUSES.**—Fire and steam play such an important part in everyday life and work that the accidents producing burns and scalds are infinite in their variety. Among the commonest are the ignition of clothes, especially those of women and children, the bursting of paraffin or other lamps, the spilling of hot fluids, molten metal, or boiling oils, explosions of gas or gunpowder, and the escape of steam in boiler explosions. Severe burns sometimes follow the application of very hot poultices to young and debilitated children.

Such substances as hot solids, molten metal, and strong caustics are local in their action, and produce burns which, although deep, are limited in extent. Flames, exploding gases, and vapours, on the other hand, occasion more widespread injury to tissues, but the damage is comparatively superficial. Then risk, however, is accentuated by the inhalation of poisonous fumes or solid particles of carbon and dust into the air-passages and lungs.

Scalds resulting from boiling water or steam are less severe locally than those caused by boiling oils or saline fluids, but the inhalation of steam into the air-passages introduces additional risks in the form of oedema glottidis, bronchitis or broncho-pneumonia, complications which may also follow burns of the mouth or pharynx resulting from drinking very hot fluids or caustic substances.

Since electricity has come to assume such a prominent place in industrial undertakings, a distinct form of burning has been recognised. Mery and Douhaquet-Labordene have studied this variety of burns, and have formulated the following propositions regarding them: (1) They may be superficial or deep, (2) they are not painful, (3) they are not accompanied by fever, or followed by any constitutional disturbance, (4) being aseptic they do not tend to suppurate, and (5) they cicatrise quickly and well.

**RISKS.**—The main factors upon which the risk of a burn depends are—(1) its *extent*. It has been shown that burns implicating from one-half to two-thirds of the entire surface of the body are almost invariably fatal. (2) Its *situation*. Burns over the serous cavities of the body—abdomen, thorax, or skull—are, *ceteris paribus*, much more dangerous than those on the limbs. (3) *The age of the patient*. Although young children succumb to the shock of severe burns more readily than adults, they appear to withstand prolonged

suppuration better. (4) The presence or absence of *sepsis* is a most important factor in regard to prognosis.

**CLASSIFICATION OF BURNS**—The classification most generally accepted is that of Dupuytren, which has for its basis the depth to which the injury has penetrated. He distinguishes six degrees—I Hyperæmia or Erythema, II Vesication, III Partial destruction of the thickness of the true skin, IV Destruction of whole thickness of the true skin, V Charring of muscles, and VI Charring of bones.

#### CLINICAL FEATURES OF BURNS

**GENERAL PHENOMENA**—The severity of a burn depends chiefly on the extent of surface implicated, and to a less degree on the depth of the lesion. The intensity of the heat and the duration of its application are also important factors.

Almost all burns which are of sufficient severity to be brought under the notice of the surgeon present in point of time three distinct clinical stages—I The stage of *congestion and pain*, II the stage of *fever and inflammation*, and III the stage of *suppuration*.

I **Stage of Congestion and Pain**—This stage lasts about forty-eight hours, during which the patient is in extreme agony, and the whole area involved in the injury is intensely congested and somewhat swollen. The patient suffers from a form of traumatic shock or collapse, attributable to reflex stimulation of the sympathetic nervous system. As a rule he lies moaning with pain, the face pale, the features drawn or shrivelled, the skin dry and moist with a cold, clammy sweat. The temperature is below the normal, the pulse small, weak, and almost imperceptible and the respirations shallow and irregular. The urine is scanty and high-colored, and the vital reactions gradually diminish. In cases which prove rapidly fatal the mind is clear at first, and may remain so to the end, or coma may supervene before death ensues. In some cases, especially in children, great mental excitement followed by delirium, cramps, and convulsions are marked clinical features before the fatal issue.

In many cases even of great severity the patient shows a wonderful degree of apathy, lying still, and complaining of nothing except great thirst.

II **Stage of Fever and Inflammation**—This stage—the stage of reaction—begins on the second or third day. The pain subsides, the appetite is lost, and constipation, or it may be dysenteric diarrhœa, is present. There is a general congestion of the internal viscera, which may give rise to various clinical complications. This *albuminuria* is very constantly present in cases of extensive burning, especially when the temperature is raised to 101° F or higher. **Respiratory complications** in the form of bronchitis, broncho-pneumonia, or pleurisy are common

sequelæ of burns, especially of those occurring over the thorax. Laryngitis and œdema glottidis not infrequently follow scalds produced by the inhalation of steam, or from drinking very hot fluids or caustics.

The **intestinal complications** vary from a slight catarrh with diarrhœa to severe degrees of inflammation and ulceration of the intestinal mucous membrane. Ulceration of the duodenum, leading to perforation, has been observed in cases of severe burning, but its frequency and clinical importance appear to have been exaggerated. It has been ascribed to the digestive action of the gastric juice on the devitalised mucous lining of the gut.

**Cerebral complications** in the form of meningitis or cerebritis are not infrequent after burns implicating the scalp and neck, and are evidenced by cerebral irritation or delirium followed by convulsions and coma.

III **Stage of Suppuration**—This stage begins when the sloughs separate, and its duration and severity largely depend upon the success which attends the efforts made to secure asepsis in the local condition. In severe cases with marked septic infection it may be very prolonged. The patient gradually loses strength, and evidences of septic absorption in the form of septicæmia or pyæmia manifest themselves. At one time tetanus was a not infrequent sequel. Should the suppuration be profuse and long-continued, waxy degeneration of the liver, kidneys, or intestines may supervene, and death result from hectic. In this stage death has been attributable in some cases to perforation of a duodenal ulcer.

**LOCAL PHENOMENA**—The description of the different degrees of burns may be prefaced with the remark, that clinical cases almost invariably illustrate more than one degree of burning. The deeper varieties are always accompanied by those of less severity, and the clinical picture is made up of the combined characteristics of all. Further, it may be said that it is often extremely difficult to determine the exact depth of a burn immediately after it occurs, sometimes it turns out less severe than it appeared at first sight, but oftener, perhaps, more so.

**BURNS OF THE FIRST DEGREE—Erythema or Erythema**—These are usually produced by flame in contact with the skin for a very short time, by solids or liquids below 100° C, or they may result from prolonged exposure to the summer sun's rays.

The pain is acute while it lasts, but it usually passes off in a few hours. The part is of a bright red or purple hue, which disappears temporarily under the pressure of the finger, and which gradually blends with the normal color of the skin around. There is a transitory swelling of the burned area, with perhaps slight oozing of serum from the surface, and the superficial layers of the cuticle usually peel off later.

**BURNS OF THE SECOND DEGREE — Vesication —** More prolonged exposure to flames, solids, or fluids at or above 100° C, or direct application of steam, are the common causes of burns of the second degree.

Here and there over an area which presents all the features of a burn of the first degree to an aggravated extent, there appear, either immediately after the accident, or, more frequently, not till some hours later, a number of vesicles, or blisters. Each vesicle consists of a raised portion of epidermis, under which may be seen serum of a yellowish or brownish colour. Sometimes the vesicles burst, the serous or sanious fluid escapes, and the surface underneath is seen to be of a bright scarlet colour, the papillae of the skin standing out as fine velvety projections, excessively sensitive to pressure or friction, and from which serous fluid freely oozes. No permanent scar remains after the healing of such burns, but the part may for some time show a slight depression or dark-coloured pigmentation. Infection by septic bacteria may induce superficial suppuration, and so delay repair.

**BURNS OF THE THIRD DEGREE — Partial Destruction of the True Skin —** This usually results from contact with hot metals, the prolonged application of flames, or from fluids at a temperature above 100° C.

The epidermis, Malpighian layers of the skin, and the papillae are more or less disorganised, and patches may be completely destroyed, leaving hard, dry, and shrunken sloughs of a yellow or black colour, and quite insensate. The surrounding areas show damage to the first and second degrees. The pain in these burns is intense, but passes off during the first day, to return again, however, when, about the sixth or seventh day, the sloughs separate and expose the nerve filaments of the underlying skin. Granulation tissue fills up the gap, and by its contraction may lead to a certain amount of depression in the pale scar which results.

**BURNS OF THE FOURTH DEGREE — Total Destruction of the True Skin —** Any form of intense heat if sufficiently long applied may produce burning to the fourth degree. The local destruction of tissue extends right through the true skin and usually involves the underlying connective tissue and fat. Large black, dry eschars are formed, around which a ring of white tissue is seen, and outside this a zone of intense congestion which gradually merges into the normal skin. As the cutaneous nerves are completely destroyed in burns of this degree, pain is not severe. Infection is very prone to occur by organisms passing from the surrounding skin into the sloughs, and profuse suppuration often ensues. Cicatrization is always slow, and leaves deep, irregular, and often puckered cicatrices, which by their contraction in course of time tend to produce varying degrees of

deformity according to their situation. In the region of the neck, the face, or the flexures of large joints the distortion is often extreme, and may call for active surgical interference to remedy it.

**BURNS OF THE FIFTH DEGREE — Charring of Muscles —** This degree of burning is frequently met with in epileptic, apoplectic, or alcoholic persons who, while unconscious, have remained for a prolonged period in contact with hot metal, flame, or chemicals. All the tissues of the part down to the muscles are destroyed, and joints or serous cavities may be opened into.

Large nerves or vascular trunks may be implicated, and profuse hemorrhage may occur, either at the time of the accident, or later when the sloughs separate by suppuration. Septic infection of any synovial or serous cavity which happens to be implicated is more than probable.

In many cases the only hope for the patient lies in amputation.

**BURNS OF THE SIXTH DEGREE — Charring of Bones —** When burning goes to this extent the result is usually fatal, and when a limb is implicated amputation is imperative.

**Pathology of Burns —** The cause of death in rapidly fatal, extensive, superficial burns is the chief point of pathological interest. It has long been recognised that the general constitutional disturbance following extensive superficial burns is proportionately much greater than is observed in severe, deep burns. As Barden puts it, "There seems to be something especially noxious in the merely superficial burn." Such injuries are almost universally fatal, especially in children, when more than one-half of the surface of the body is involved, even should the burns be of the most superficial kind. Many theories have from time to time been put forward to explain this fact. Thus the views that the grave symptoms resulting so often in death are due to interference with the respiratory, the excretory, the heat-regulating, or even the protective functions of the skin, have each had their advocates. More extended observation by modern methods, however, has thrown doubt upon most of these theories.

The chief naked-eye lesions found after death have been a general hyperæmia of the organs of the abdominal, thoracic, and cerebro-spinal cavities, sometimes accompanied by serous or sanious exudation — conditions which have been attributed to a reflex vasomotor disturbance.

These changes appear to take some time to be produced, because if death be very rapid they are not observed. If the patient survive a few days, active inflammatory signs may supervene in the intestinal canal, lungs, pleura, kidneys, or meninges, and ulceration may follow in the stomach or duodenum. The relative frequency of these lesions may be gathered from the following statistics of 200 cases by Schjerning —

Hyperæmia of brain	occurred in 49	p.c.
Hyperæmia of lungs	" "	36.5 "
Nephritis	" "	28.2 "
Pneumonia	" "	27 "
Hyperæmia of intestines	" "	22.2 "
Pleurisy	" "	18.8 "
Ulcer of duodenum	" "	12.4 "
Meningitis	" "	10.5 "

There is also a profound alteration of the blood in these cases. Its specific gravity is increased, the polymorpho-nucleo leucocytes are increased in number, the red cells run together and ultimately break down, and the hæmoglobin set free is deposited in the epithelial cells of the tubules of the kidney, where it produces irritation followed by hæmoglobinaemia.

Thrombi and small extravasations are found in many situations throughout the body, probably due to the action of a fibrin-ferment, of which two have been isolated, one a body like murex, the other belonging to the pyridine-choline group.

Some interesting clinical and experimental observations by Korolenko throw light on these morbid appearances. He found both in patients and animals dying after severe superficial burns, that the ganglion cells of the solar plexus had undergone degenerative changes, varying according to the severity of the injury, from slight oedema to complete destruction of their protoplasm with disappearance of their nuclei. From his experiments he concludes that the solar plexus is subjected to a reflex excitation, which passes out to the abdominal circulatory system, producing spasm of the vessels, including those supplying the ganglia themselves, whereby nutrition suffers. In the rapidly fatal cases the vessels remain spasmodically contracted, and hyperæmia of the abdominal organs is not observed. In the less severe cases the spasm of the vessels is followed by a passive dilatation which explains the hyperæmia of the viscera. The influence of the solar plexus upon the heart may also be a factor in producing death.

Bardeen has recently observed marked changes in all the lymphatic tissues of the body, from the small nodules of lymphocytes in Glisson's capsule to the largest lymphatic glands consisting in general oedema of the lymph tissue, especially at the germinal centres, with swelling of the individual cells and breaking up of their nuclei, and the appearance of certain large flattened endothelial cells having a phagocytic action. He also found in his cases marked cloudy swelling of the liver and kidney, and softening and enlargement of the spleen.

The morbid findings throughout, therefore, are so like those met with in diseases where the presence of a *toxin* is known to produce them, that it seems highly probable that death in rapidly fatal cases of burning admits of a similar explanation, although the source and nature of the toxin are as yet undetermined.

**Local Histological Changes.**—The most noteworthy changes following the application of heat to the surface of the body are the coagulation of the protoplasm of the cells, and its subsequent disorganisation. The collagenous bundles in the immediate vicinity of the charred tissues become swollen and thickened, and large irregular spaces are formed by the sudden formation of steam in the tissues. The papillæ are swollen, thickened, and spherical, and become loosened from the epidermis. By the over-stretching of the horny layer of the skin and the sudden evolution of steam, the epidermis is raised, and bulle form. Into these serum, sometimes mixed with blood, is effused. The blood-vessels and lymphatics of the tissues are seared and occluded. The zone surrounding the destroyed tissue is congested and swollen, and shows considerable infiltration with leucocytes. The separation of eschars is effected by the growth of granulation tissue.

#### TREATMENT

**OF THE GENERAL CONDITION.**—During the stage of collapse or shock the patient must be kept warm by means of hot bottles, warm blankets, or cotton-wool. Alcoholic stimulants, either by the mouth or as an enema, are clearly indicated, and should be given freely. Hypodermic injections of ether, strychnine, or brandy may be called for.

When pain is severe, morphia must be injected hypodermically.

As improvement takes place, hot drinks will do good if the patient can take them.

**LOCAL TREATMENT.**—It cannot be too strongly urged that burns must be treated on the same rigidly antiseptic lines as other surgical wounds. In this connection it is important to note that Uma has invariably found pyogenic bacteria in the bulle of burns.

The first step, therefore, is thorough purification by antiseptic agents, and that this may be satisfactorily accomplished a general anæsthetic is in most cases necessary. In the subsequent dressings of extensive burns, especially in children, the value of a general anæsthetic can scarcely be over-estimated. The clothes are carefully removed, the burned area thoroughly, yet gently, washed with warm boracic lotion or a 1 in 100 solution of lysol. Blisters are opened and the raised epithelium removed, and a final wash with normal salt solution, to get rid of any excess of antiseptic, completes the preliminary purification.

The local application to be used depends largely upon the depth of the burn, its extent, and the time which has elapsed since its occurrence. In all cases the following indications have to be met—(1) the relief of pain, (2) the prevention of sepsis, and (3) the promotion of cicatrization.

**LOCAL TREATMENT OF RECENT BURNS OF FIRST,**

**SECOND, THIRD, AND FOURTH DEGREES—Picric Acid.**—Within recent years—thanks to Thiercy and others of the French school—we have found in *picric acid* an agent which meets all our requirements in a way not previously attained by the older methods. It is employed in solutions varying in strength from 1 in 100, which is practically a saturated watery solution, to 1 in 50 when alcohol is added. It may be dissolved in sulphuric ether forming a solution of about 1 in 20, which is painted on to the affected area, and on the evaporation of the ether leaves a covering of a very fine powder of picric acid.

A useful lotion of moderate strength is made as follows—

Take of Picric acid	1½ drachm
Absolute alcohol	3 ounces
Distilled water to	40 ounces
Dissolve	

After thorough purification of the burned area by antiseptic lotions, pads of lint or sterilised gauze are lightly wrung out of this solution, and applied over all the affected surface and for some distance beyond. A moderately thick layer of antiseptic wool is put on over this and retained in position by a bandage, preferably a many-tailed bandage, to obviate the necessity for much movement during dressings. A splint to ensure rest is advantageous when possible. It is important to observe that the dressing is applied *without* any waterproof covering—that is to say, it is not in the form of a poultice.

This dressing may be left in position for from three to seven days, according to the severity of the burn and the degree of asepticity. In burns of the first and second degrees it will be found that in three or four days all will be healed under a single application. In deeper burns, especially when the sepsis may not be absolute, it is well to change the dressing on the third or fourth day. Any portion of the original dressing which remains perfectly dry and adherent to the surface need not be removed, but should simply be moistened by pouring a stream of picric acid lotion over it. Any parts of the dressing which are moist from discharge must be removed, the surface thoroughly purified with boracic acid or lysol, and the picric acid pads reapplied. The dressing should be repeated once or twice a week according to circumstances.

Among the *advantages* of the picric acid method may be mentioned its simplicity and safety, as well as the infrequency with which dressings have to be changed. In a very few cases it has been found to cause temporary pain, but in the vast majority it is not only painless but even anodyne. Although not a powerful antiseptic it is sufficiently strong to maintain the asepticity of a burn which has been carefully purified at the outset. Its absolute dryness is an important factor in preventing the develop-

ment of septic bacteria. But its most valuable feature as a dressing for burns is the marked power it has of promoting the proliferation of epithelium—its *keratoplastic action*—which seems to depend upon the coagulation of the albuminous exudate from the injured surface forming a non-irritating, aseptic protection to the young epithelial cells.

Its only *disadvantages* are that it temporarily stains the skin of the patient, and the hands of the surgeon and nurses. This to a large extent may be prevented by smearing the hands with vaseline before using it, and afterwards washing in methylated spirit or turpentine. Stains on cotton or linen clothes are readily removed by ordinary laundry processes, but remain permanent in woollen and flannel articles.

In a few isolated cases toxic symptoms—nausea, vomiting, diarrhoea, dark-coloured urine, yellowness of skin, and drowsiness—have been alleged to follow its use, but those who have had most experience with picric acid have not observed any symptoms attributable to its absorption. The writer, after an experience of over two hundred and fifty cases treated by this method, has not yet met with any untoward effects. In young children the solution may be diluted to about half its strength with advantage.

The *results* are best in superficial burns of the first, second, and third degrees, but the agent is useful as a primary dressing in burns of all degrees. After the sloughs have separated and a granulating surface is left, the usual applications for an aseptic healing sore should be substituted. When the area is large and cicatrization slow, recourse should be had to skin-grafting by Thiersch's or one of the other methods available.

**Ichthylol and Thiol.**—These, as antiseptics and keratoplastics, are inferior to picric acid. They both cause considerable pain when first applied, but this soon passes off. Ichthylol is applied as a 30 per cent solution in water, thiol, either in watery solution 1 in 4, or as a powder, mixed with subnitrate of bismuth and a small quantity of iodoform.

**Aseptic Treatment.**—Mme Nageotte has advocated the treatment of burns by simple aseptic dressings after thorough preliminary purification with chemical antiseptics. The results have not been entirely satisfactory, doubtless from the difficulty of obtaining absolute asepsis, and from the absence of any agent active in the promotion of epithelial regeneration.

**Moist Applications.**—Gummy substances, like carron oil, carbolic oil, and boracic ointment, substances calculated to occlude the burn, like collodion, dry powders, etc., are only mentioned to be condemned. They entirely fail to meet the indications for the rational treatment of burns on modern lines, and should be abandoned.

**TREATMENT OF COMPLICATIONS.**—Renal, pul-

monary, cerebral, and other clinical complications of burns are treated on the same lines as similar conditions arising from other causes, and do not call for further mention here.

**TREATMENT OF THE RESULTS OF BURNS.—Ulcers.**—After the sloughs have separated from a burn and a stage of suppuration has been reached, an ulcerated surface is left which must be treated on the principles governing the management of ulcers. In the covering-in of raw areas left by burns, skin-grafting finds one of its most useful applications. Means should be taken to prevent the production of deformities by the contracting scars during the healing of these ulcers.

**Cicatrices.**—The cicatrices following burns often constitute serious disfigurements, many of which may be amended by plastic surgery. They are also particularly prone to the various diseases of cicatrices (*q v*).

**Deformities.**—The contraction of extensive cicatrices, especially in the region of the face, neck, and flexures of joints, often leads to considerable deformity and interference with function, for which plastic operations may do good.

#### MEDICO-LEGAL ASPECTS

For medico-legal inquiries the following points may be noted:—

**The Mode of Production.**—Moist heat in the form of boiling water or steam leaves a widespread, red, soft, and sodden condition of the skin and underlying parts, without any actual destruction of tissues. Dry heat from molten metal, red-hot solids, resins, etc., results in a more limited damage of the tissues, which will be blackened, dry, and more or less completely destroyed by charring. The damage done by boiling oils resembles that from dry heat more than from moist, while flames produce injuries simulating both. The hairs of a part are singed by dry heat, but remain intact after the application of moist heat.

**The Results of Explosions.**—The injuries resulting from explosions of gas are of wide extent and superficial, and consist chiefly in scorching of the surface. In gunpowder explosions there are numerous small particles of carbon embedded in the skin.

**Was the person alive or dead when burned?**—In conflagrations persons are often suffocated by smoke, gases, or dust without being burned. In other cases they may be killed by suffocation and then burned. If there are vesicles on the skin, surrounded by a red and swollen area, and containing serous or sanguous fluid, the presumption is that the person was alive when burned. The absence of these signs, however, does not negative the life of the person when subjected to the fire, as they often take some hours to appear, and death may have ensued before they had time to form. The skin under a vesicle produced during life is intensely injected, while that on a body burned after death

is hard, dry, and yellow, and any blister which may have formed only contains gas. It is to be kept in mind, however, that heat applied to a dead body while still warm produces appearances closely resembling those of burns inflicted during life. The main difference is that the fluid in the vesicles in the former case is thin and watery, and contains a very small proportion of albumen.

In burns produced by red-hot solids during life the skin round the destroyed tissue is usually white, with a deep red ring beyond it. In a dead body this red area is absent, and the whole of the destroyed surface is deadly white.

**How long did the person survive the burning?**—This question will be answered by noting whether there are present only the signs of actual burning, or whether advanced inflammatory signs have supervened. Suppuration, the separation of sloughs, the formation of granulation tissue, and the onset of gangrene indicate a considerable lapse of time.

**Marks of Violence on a Burned Body.**—The body may have been burned to conceal marks of violence. Careful examination may reveal such marks, unless the destruction be too complete.

**Spontaneous Combustion.**—It is almost needless to say that the belief that spontaneous combustion may take place in the human body can no longer be maintained.

**Burns, Diameter of.**—The diameter measured from the promontory of the sacrum to the crest of the pubes, above the obturator foramen, the sacro-pectineal diameter.

**Burquism.**—Met illotherapy, Perkinism, tractionation. *See Hysteria (Treatment by Suggestion)*.

#### Bursæ, Injuries and Diseases of.

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*See also* ELBOW-JOINT, INJURIES AND DISEASES (*Bursitis*), GANGLION (*Diagnosis*), HIP-JOINT, INJURIES (*Bursal Enlargements*), HIP-JOINT, DISEASES (*Bursa*), KNEE-JOINT, DISEASES (*Bursa*), SHOULDER, DISEASES AND INJURIES (*Bursal Enlargements*), SYPHILIS (*Bursæ*).

**INTRODUCTION.**—Two varieties of Bursæ are found, the true and the false. A true bursa is



constant, while a false bursa appears adventitiously over some abnormal prominence of bone, *eg* the so-called bunion, or the thickening over the prominent spinous processes in an angular deformity of the spine. The size of a false bursa is a fair indication of the duration of the abnormal prominence of bone, and the longer the false bursa has existed, the nearer does it approach in structure to the true bursa. A true bursa is a simple synovial sac placed between two surfaces to prevent friction, and such sacs are either deep-seated or subcutaneous.

In structure a bursa is a sac consisting externally of areolar tissue of varying density, and lined internally by a more or less perfect synovial membrane of flattened cells. The cells are not continuous over the whole surface of the synovial membrane, but are distributed in patches, the spaces between being filled by the ground substance of the connective tissue of the wall. Numerous blood-vessels and some nerves are found in the walls, and when enlargement takes place the vessels increase in size and number. The cavity of a bursa is a lymphatic space in the same sense as is the synovial cavity of a joint, in fact, a bursal cavity is an isolated articular cavity. The false bursæ are at first recesses in the subcutaneous connective tissue, and are not bounded by true synovial membrane, but after a time patches of cells which have an epitheloid appearance are seen in the walls, and a more or less perfect bursal cavity is formed. The synovial sheaths of tendons are true bursæ, and facilitate the movements of the tendons in their osseo-fibrous grooves. These sheaths in no way differ in structure from bursæ.

**SITUATIONS OF BURSA.**—In the *head, neck, and trunk*. Apart from the intra-articular synovial sacs of the temporo-maxillary, vertebral, and intercostal articulations, bursæ are very few in number. An important one is interposed between the posterior surface of the body of the hyoid bone and the thyro-hyoid ligament. It sometimes becomes enlarged and forms an encysted fluid swelling, projecting in the floor of the mouth.

In the *upper extremity* bursæ are very numerous. An important one surgically is the large subscapular bursa between the subscapularis and the deeper muscles. This bursa is often the site of the dry form of bursitis, when movements of the scapula become painful, limited, and accompanied by distinct creaking. If fluid is poured out the scapula is displaced backwards. A large subcutaneous and often false bursa is found over the acromion process of the scapula, especially in those who carry weights on their shoulders. In the immediate neighbourhood of the shoulder-joint are three bursæ, one beneath the tendon of the subscapularis, which frequently communicates with the joint, one beneath the tendon of the infra-spinatus, which rarely opens into the joint, and

one large sac beneath the deltoid, which has no direct connection with the joint. The investing sheath of the long head of the biceps may be regarded as a bursa. Between the tendon of the *teres minor* and the shoulder a bursa is occasionally found, and one between the tendons of the *teres major* and *latissimus dorsi*. In the neighbourhood of the elbow-joint are three well-known bursæ, one between the skin and olecranon process, a second between the tendon of the triceps and the upper part of the olecranon, and a third intervening between the biceps tendon and the tuberosity of the radius. At the wrist the tendons of both the superficial and deep flexors, as well as the median nerve, are surrounded beneath the annular ligaments by a large loose synovial membrane which extends upwards to the radio-carpal articulation and downwards to a little beyond the bases of the metacarpal bones, being prolonged farther down in the tendons of the little finger than in the others. A sheath exists around the tendon of the flexor longus pollicis as it passes beneath the annular ligament and communicates with the large flexor bursa at the upper border of the annular ligament. The large bursa of the palm of the hand is important as the site of the so-called "compound" ganglion or tubercular form of bursitis. Beneath the insertions of the following muscles bursæ are found—*extensor carpi radialis longior* and *brevior*, also between the surface of the former muscle and the *supinator brevis*. False bursæ also form over the knuckles and interphalangeal joints in those who labour manually.

In the *pelvis and lower extremity* the bursa over the tuber ischi is large and multilocular, and frequently enlarges in tailors and weavers, hence originates the expression "weaver's bottom." Beneath the gluteus maximus are two bursæ, one multilocular between it and the great trochanter, and one between the muscles and the vastus externus. Other bursæ are found beneath the insertions of the gluteus medius and minimus, while between the obturator internus and the margin of the small sciatic foramen is a bursa which is often continuous with another between the tendon of the muscle and the hip-joint. At the front of the joint is a bursa beneath the psoas which often communicates with the joint. Above the knee there is a bursa beneath the quadriceps extensor, also in communication with the joint. The bursæ in relation with the patella and its ligament are three in number. In front of the lower half of the patella and upper half of the ligament is the pre-patellar bursa of "housemaid's knee." Over the lower half of the ligament is a second bursa, and between the ligamentum patellæ and the tubercle is a third bursa. In the popliteal space are the following bursæ. On the outer side and above the joint are found one beneath the outer head of the gastrocnemius, and one beneath the tendon of

the popliteus, which is almost always an extension from the first. On the outer side below the joint are bursæ between the tendons of the popliteus and biceps and the external lateral ligament. On the inner side there is a bursa between the inner head of the gastrocnemius and the femur, which is prolonged between that muscle and the semi-membranosus, and often communicates with the joint. There is also a bursa between the semi-membranosus and the head of the tibia which is frequently enlarged, and sometimes a bursa between the tendons of the semi-membranosus and semi-tendinosus. A bursa also separates the tendons of the three inner ham-strings from the internal lateral ligament, and is prolonged beneath the insertion of the sartorius. In the foot, beneath the insertions of the tibialis anticus and sometimes of the peroneus brevis a bursa is found. One important bursa is found between the upper part of the posterior surfaces of the os calcis and the tendo Achillis. It is frequently inflamed, and the affection so constituted is called "Achillodynia."

**INJURIES OF BURSA.**—These structures, when in exposed situations, are particularly liable to traumatic either of accidental and violent character, or of a frequently recurring nature, and from the latter originates the condition known as chronic bursitis. A blow in the neighbourhood of a bursa results either in a contusion around the bursa or hemorrhage into the bursal cavity from rupture of the vessels supplying it, an event especially liable to occur in a hemophilic patient, or the bursa is ruptured, a very probable event if it already contains fluid, or it may be cleanly torn open or lacerated. Injuries not sufficient to cause severe hemorrhage are followed by acute serous effusion. But if hemorrhage occur it may either be absorbed or sometimes followed by suppuration, or failing to be absorbed it results in a fibroid bursa. Blood may be effused in such large quantities as to mask a severe injury of the bone beneath, *e.g.* fracture of the patella or of the olecranon.

The treatment is as follows.—If there is no abrasion of the surface a cooling application should be employed for the first thirty-six hours and the part kept at rest. Then hot applications may be used to aid absorption, assisted by pressure and gentle friction. Should the part become acutely inflamed, it is better to make a free incision, for the bursal sac is a lymph space and absorption from it readily occurs. If the bursa is wounded or lacerated it should be thoroughly cleansed with antiseptic fluids, and a gauze drain placed in the cavity for two days.

**BURSITIS.**—(a) *The Simple Acute Form (Acute Hygroma)* is etiologically of two varieties, Traumatic or Primary, and Secondary from extension of inflammation in the neighbourhood. Trau-

matic hygroma follows injuries, especially contusions and penetrating wounds, if it occur in bursa already inflamed, suppuration often ensues, and hygroma is occasionally seen from excessive over-use. The secondary form is due to extension to the bursal sac of inflammation in the vicinity, although the sac may not be in direct communication with the part. Acute secondary bursitis is therefore seen in connection with a boil, carbuncle, erysipelas, suppurative arthritis. The secondary form is either serous or suppurative, and the latter is either localised or diffuse. The serous form (acute serous hygroma) is accompanied by phenomena of gravity such as very acute pain, redness, œdema, and the formation of a tumour often within the first twenty hours. If the effused fluid remains serous, the swelling lessens in five or six days, and the inflammatory symptoms subside, and the hygroma either disappears or becomes chronic, or pus may form. Should the suppuration be localised to the bursa, it will, if left to itself, burst, either externally and be followed by the formation of two or three fistulous openings, after which the bursa may shrink and disappear or it will invade the neighbouring tissues and cause extensive cellulitis, and we have seen a suppurating bursa patellæ followed by necrosis of that bone and by acute arthritis. The treatment is evidently to open the inflamed bursa freely so soon as any suspicion of pus arises.

(b) *Simple Chronic Bursitis (Chronic Hygroma).*—**Etiology.** It frequently follows acute, but more often is the result of occupation, as in housemaids, cobblers, tailors, and nurses. Verneuil describes bursa as existing over tumours, and Cruveilhier met with a large serous bursa between a mammary scirrhous and the pectoralis major. Pre- and peri-hepatic cysts are often bursal in their nature. False bursæ are met with over the prominences in club-foot and in bunions. The varieties of chronic hygromata are the cystic, proliferating, fibrous, and hemorrhagic. A cystic hygroma is the most frequent, and corresponds pathologically to hydrothorax. It is sometimes bi-lobed in shape, but is usually unilocular, rarely multilocular. The wall is composed of thickened fibrous tissue, cartilaginous in places, or incrustated with calcareous plates, and the bursa often lies in a fatty envelope, *(cf. hygroma lipomatosa de la nuque in porters)*. The contents are either clear or opaque fluid, of yellowish colour, varying in consistence and containing many cholesterol crystals. Sometimes, however, it is bloody or purulent. In the wall there are often large arteries present, which may give rise to hemorrhage. The proliferating hygroma is generally unilocular. The cavity contains a variable quantity of liquid, and has loose bodies floating in it which have been detached from the interior of the sac. On the latter are seen numerous

vegetation-like bodies which are wart-like in appearance, sessile or pedunculated, and composed of fibrous tissue infiltrated with cartilage, and sometimes with uric acid or phosphate of lime. The fibrous hygroma is met with, especially in front of the knee, as a firm, indurated mass, in the centre of which is found a little fluid. The hemorrhagic hygroma is either the result of traumatism, or the blood may be spontaneously effused. The fluid in the cavity is thick and black, or may consist almost entirely of clotted blood. Volkmann has described a parallel condition in joints as "pachysynovitis hemorrhagica."

The symptoms of chronic bursitis are not difficult of recognition. In an appropriate situation a small rounded or flattened tumour commences which is indolent, nearly painless, fluctuating, and on pressure may be rolled under the hand, giving a feeling of fine crepitation. A fibrous hygroma is from the first hard and unyielding in character, while a hemorrhagic hygroma has very few distinguishing characteristics. The course of a hygroma is chronic. It may disappear entirely, or it may undergo progressive sclerosis and contraction of its walls. The chronically enlarged bursa is exposed to rupture, hemorrhagic effusion, and suppuration. If it be subcutaneously ruptured the fluid is absorbed by the tissues, but the bursa reappears.

*Treatment* — There are practically two methods of treatment — obliteration and excision of the sac. In the early stages of chronic bursitis, if the irritating cause be removed, the bursa will often subside, only to reappear when the cause is at work again. Counter-irritants are sometimes of temporary service. The means adopted for obliteration of the sac are incision, and the application of nearly pure carbolic acid, or simple incision and stuffing the cavity with gauze, so permitting it to granulate up entirely. But in all forms of chronic bursitis of long standing, especially the proliferating and fibrous, complete removal of the sac is necessary.

(c) *Gouty, Gonorrheal, and Rheumatic Bursitis* — Allusion has already been made to the deposit of urate of soda in the walls of bursa, and if the subject be affected with well-marked gout, this salt is often deposited continuously over the whole interior of the sac and in its walls. The bursa most frequently affected are the olecranon, patellar, and the subcutaneous bursa of the hands and feet. In gonorrheal and acute rheumatism the bursa in the neighbourhood of an inflamed joint often sympathise acutely. In the early stages a dry hygroma is present, which is painful and finely crepitating to the touch, but serous effusion sets in to a varying degree, and the bursa swells somewhat. Its symptoms then approach those of the serous variety of acute bursitis. The diagnosis is made and treatment carried out by recognising the associated conditions.

(d) *Tuberculous Bursitis* — This was formerly thought to be rare, but many cases which were published under the title of fungous disease of the bursa are now recognised as tuberculous bursitis. The sites of the affection are the olecranon, subdeltoid, sub-scapular, pre- and infra-patellar, malleolar, psous and popliteal bursa.

Two varieties of tuberculous bursitis are met with, the primary and the secondary. Of the existence of the former there can be no doubt, and all conditions are present in bursa for the development of tubercle in suitable subjects, such conditions being exposure to injury, abundant fibrous tissue, and the peculiar synovial character of the lining wall of the sac. Secondary bursitis is distinguished from primary by its being associated with tubercle of the neighbouring joint. It is not essential that there should be any direct communication between the synovial membranes of the bursa and joint. Pathologically three forms are recognisable — fungous hygroma, hygroma with rice-grain bodies, and mycetozoa or mucoid hygroma. Of these the fungous hygroma is most common in type. At first the cyst is simply serous and its wall is not thickened, but it soon becomes covered by small tubercles. This stage often escapes observation, but later granulation tissue forms and caseates, and pus is seen, so that the general appearance of the cavity is like that of tuberculous synovitis. It is probably a slow form of tubercle which affects bursa, as the results of inoculation of animals take a long time to appear. In the rice-grain-bodied form of hygroma there is but little fluid. A large number of these curious bodies is present, and that they are truly tuberculous is shown by the results of inoculation (Nicaise, *Revue de chir.*, 1885). This form of tuberculous bursitis is frequently met with in the large palmar bursa, and used to be known as "compound ganglion." The mucoid form of tuberculous hygroma has been described by Citzmann (*Médecine moderne*, 1890, p. 638). There is found a cyst filled with gelatinous material, translucent and red or yellow in colour. The nature of this material has been conclusively proved by inoculation. The symptoms are those of a slow chronic swelling, often thick-walled and giving the impression of a solid bursa as the fluctuation is obscured. In the rice-grain form fine crepitations are present. Unlike other bursa, the more tuberculous a bursa grows, the softer and more semi-fluctuating does it become. This is a diagnostic sign of great importance. Eventually a fistula forms, but before that has occurred, extension has taken place to bones and joints. The only permissible treatment is extirpation.

(e) *Syphilitic Bursitis* — It is met with in the secondary stage of syphilis, and as the subcutaneous form, and in the tertiary stage as the fibroid variety which becomes gummatous. The latter variety may occur as long as twenty years

after the primary infection, and frequently follows an injury. The seat of election is the patellar bursa. The progress of a tertiary syphilitic bursa is very slow, but ultimately the tumour becomes adherent to bone or skin and ulcerates. The diagnosis is often difficult, as it may be mistaken for simple chronic hygroma, old hæmatoma or nodes, and when it ulcerates it may simulate tubercle. It is, however, very indolent and slow in progress, but when small readily yields to treatment. If of large size or ulcerating it should be completely removed by excision.

**NEOPLASMS**.—The innocent forms met with are fibro-enchondroma and myxoma. The latter has been seen by Chevré three times. In one case the tumour had attained the size of a child's head and was freely ulcerated on the surface. Fibro-enchondroma is comparatively rare. Considerable difficulty is met with in diagnosing these conditions, and the removal is frequently undertaken, not because their nature has been recognized, but because of their size. Malignant new growths are either sarcomata or endotheliomata. The sarcoma is either of the soft or round-celled type, or is of the rapidly-breaking-down hemorrhagic variety. With reference to endothelioma, Dallinger has met with one case in a fistulous bursa of a woman aged 58, and Mikulicz has seen a similar case. The softer varieties of neoplasia may attain an enormous size, and Ranke (*Archiv für die Clin* 1886) has met with one 62 cm in circumference. The treatment of these neoplasms is complete removal.

**Bursinic Acid**.—So named from *Bursæ pastoris* (shepherd's purse), from which it is obtained, it is believed to act like ergotine.

**Bursitis**. See BURSÆ, INJURIES AND DISEASES OF.

**Bushman**. See ETHNOLOGY.

**Bussorah Boli**. See FURUNCULUS ORIENTALIS.

**Butane**.—The name given to two isomeric hydrocarbons of the paraffin series, which are colourless, inflammable gases. (1) normal butane (butyl hydride, diethyl, propylmethyl) is  $\text{CH}_3\text{CH}_2\text{CH}_2\text{CH}_3$ , and (2) isobutane (isobutyl hydride, trimethylmethane) is  $\text{CH}_3\text{C}(\text{CH}_3)_2$ .

**Butcher's Pemphigus**.—Pemphigus acutis malignus, a disease occurring in those who are constantly handling meat. See PEMPHIGUS.

**Butea Semina**.—Butea seeds, from the *Butea frondosa* or dhak-tree, are official in the Indian and Colonial Addendum (1900) to the British Pharmacopœia of 1898, in the form of *Pulvis Butea Seminum*, they are given internally in place of sanantonin (dose, 10 to 20

grains), externally, they are used for a ring-worm paste, the oil obtained from the seeds is called moolooga oil.

**Butter**. See DIET (*Milk and its Products*).

—(1) The fatty substance obtained from cream by churning, it contains casein, lactose, salts (phosphates), water, and various fats (glycerides of palmitic, stearic, oleic, butyric, and caproic acids), along with an aromatic principle. (2) The name is also given to butter-like substances, such as alien butter (the exudation from the African butter-tree), butter of cacao, butterine, oleomargarine, etc.

**Butter Bacillus**.—*Mycobacterium butyri*. See TUBERCULOSIS (*Diagnosis of Bacillus*).

**Butterfly Lupus**.—Lupus erythematosus affecting the bridge of the nose and the cheeks. See LUPUS ERYTHEMATOSUS.

**Butterfly Operation**.—A form of posterior colporrhaphy. See UTERUS, DISPLACEMENTS (*Descent*).

**Butterfly Pessary**.—An expanding uterine support, not now much used, Zwanck's pessary.

**Buttermilk**. See INVALID FEEDING (*Milk*).

**Butter of Antimony**.—Antimony chloride, used in the preparation of antimomous oxide, a poison. See ANTIMONY, TOXICOLOGY (*Antimony*).

**Buttocks**. See BUTT-SORES, RECTUM, DISEASES (*Structure*).

**Button**.—A small knob or disc, such as the canterly button, or Corrigan's button, or, more specially, J. B. Murphy's button (a device used in connection with the operation for the establishment of intestinal anastomosis, to maintain the patency of the canal during healing), the name is also given to button-like structures, pathological or normal (e.g. Bickia button or Button de Ciete, and the umbilicus). *Button scurvy* seems to have been allied to syphilis (see VENEREAL DISEASE, *Allied Diseases*).

**Butyl Alcohol**.—The radicle *Butyl* ( $\text{C}_4\text{H}_9$ ) gives rise, in combination, to a number of derivatives, including *Butyl Alcohol* ( $\text{C}_4\text{H}_9\text{O}$ ), *Butyl Aldehyde* ( $\text{C}_4\text{H}_8\text{O}$ ), *Butyl Bromide* ( $\text{C}_4\text{H}_9\text{Br}$ ), *Butyl Chloride* ( $\text{C}_4\text{H}_9\text{Cl}$ ), *Chlorotone* (hypnotic) is a trichloro derivative of butyl alcohol ( $\text{C}_4\text{H}_7\text{Cl}_3\text{O}$ ).

**Butyl Chloral Hydrate**.—Butyl Chloral Hydrate, wrongly named croton chloral hydrate,  $\text{C}_4\text{H}_9\text{ClCH}(\text{OH})_2$ , is prepared by passing dry chlorine gas through aldehyde, the butyl chloral thus formed is separated and water is added, it resembles chloral hydrate in

its action, and has been used specially for neuralgia of the fifth nerve (dose, 5 to 20 grains); it dissolves freely in spirit.

**Butylene.**—One of the olefines, formed in the dry distillation of coal, etc. ( $C_4H_8$ ).

**Butyphus.**—The cattle plague

**Butyric Acid.** See MICRO-ORGANISMS (Fermentations).—This acid ( $C_4H_8O_2$ ) is formed during the oxidation of butyl alcohol (e.g. during the fermentation of cheese); it has been used as a hyptic.

**Butyrometer and Butyroscope.**

—Instruments for determining the amount of fatty matter in milk.

**Buxine.**—An alkaloid obtained from *Buxus sempervirens*, and probably identical with *Belerine* ( $C_{15}H_{21}NO_3$ ); buxidin is found with it.

**Buxton.** See BALNEOLOGY (Great Britain); HYDROPATHY, THERAPEUTICS, HEALTH RESORTS (English).

**Bynin.**—A proprietary liquid extract of malt.

**Bynol.**—An emulsion of cod-liver oil in extract of malt.

**Byres.** See MILK (Cow-sheds).

**Byrolin.**—A mixture of lanoline, glycerine, and boric acid.

**Byssinosis.**—A pneumonokomosis, due to inhalation of cotton fibre; byssophthisis. (See LUNGS, PNEUMOKONIOSIS).

**Cac- and Caco-.**—In compound words “cac-” and “caco-” signify bad, ill-conditioned, evil, or morbid. Thus *cacæmia* is a morbid or depraved state of the blood; *cacæsthesia* is an unpleasant sensation; *cacæcholia* is a depraved state of the bile, and *cacæchylia*, of the chyle; *cacæchymia* is a morbid state of the humours; *cacæcolpitis* is vulvar gangrene, *cacæcolpitis* is gangrene of the tongue, *cacæpneumonia* is gangrene of the lung, and *cacæstoma* is gangrene of the mouth; *cacængelia* is a morbid state of a nail, and *cacæspermia*, of the semen; etc., etc. See also *infra*.

**Cacao Butter.**—A concrete oil (*Oleum Theobromatis*) got from the seeds of the chocolate-tree (*Theobroma cacao*) of the Natural Order of the Sterculiaceæ. By varying the process of preparation, cocoa and chocolate are obtained. Cacao butter is used in the making of suppositories (except those of glycerine); it contains chiefly stearin, some olein, and the alkaloid theobromine.

**Caccagogue.** See APERIENTS, PURGATIVES.

**Cachets.**—Wafer papers for concealing the taste of nauseous drugs.

**Cachexia.** See also GOIT (Chronic and Irregular); MALARIA (*Sequelæ*); MAMMARY GLAND, DISEASES OF (*Carcinoma*); UNCONSCIOUSNESS (*Auto-intoxications*, *Cachexia strumipriva*).—This term, derived from the Greek words *kakos* and *êxis*, and meaning literally a bad habit of body, is one which has been used from the very beginnings of medicine. It never has had, however, a very precise meaning, and the following is its definition as given by Copeland:—“Depravity of the constitution, without fever, affecting more or less the solids, the circulating fluids, and the secretions.” In this way cachexia is to be regarded as meaning the outward and mainly the facial expression and characteristics resulting from the profound and complex changes in nutrition induced by a malady of some chronicity. It is to be noticed that cachexia is to be distinguished from diathesis, by which is meant a constitutional disposition in virtue of which an individual is liable to certain local affections of the same nature. It is also to be distinguished from the malady acquired as the result of some innate weakness or unhealthy surroundings, and no less from the conjoined symptoms and signs of the disease induced by such.

With the diathesis, with the conjoined symptoms, and with the malady itself, it has, however, frequently been confounded, and its distinction as a term from marasmus, dyscæmia, and many others, is not well marked.

There is no doubt that as our knowledge of healthy and diseased processes is becoming more exact, the term cachexia is becoming less and less made use of. In olden times the cachexias and the fevers shared a great part of the physician's attention, whilst now, the term mentioned in a text-book, is seldom deemed of sufficient importance to merit a place in the index. For this cause our more precise knowledge of disease, and our clearer grasp of the association of symptoms, form, as has been said, the explanation. The terms cachectic angina and cachectic liver abscess, of a previous generation, are hardly ever used now, the sore throat and liver abscess being ascribed to infective organisms acting on an individual of lowered nutritive power. Further, the scorbutic cachexia, the syphilitic cachexia, the cardiac cachexia of the French writers, and many others, are recognised now rather as a more or less definite association of certain symptoms or signs. It follows, therefore, that although in reality every disease has its cachexia, yet the cachexias which require detailed consideration at the present time are only three in number, viz. the cancerous, the malarial, and the metallic.

**Cancerous Cachexia.**—By this is indicated the facial aspect and the general appearance of feebleness and loss of flesh met with in individuals who have been for some time suffering from cancerous disease. In its marked forms the face is pale with a yellow or greenish-yellow tint, the cheeks hollow, the nose pointed, and the eyes sunken. Corresponding with the emaciation and muscular weakness the movements of such patients are slow, and as the result of the more or less constant pain, and of the general organic sensation of unwellness and feebleness, the expression is sad and gloomy and betokens suffering. The colour of the skin in such cases is due to changes in the blood and skin pigment, and these in their turn are probably brought about by the resorption into the blood of the fluid secretion of the cancer cells. It is to be remembered, on the one hand, that the cancerous cachexia may be closely simulated by other morbid conditions, and, on the other, that cancer may be present and yet cause no cachexia. This latter might be explained on the ground that little or no pain is present, and that no resorption is taking place from the diseased mass. It would seem that the cachexia is usually most evident in cases of ulcerating cancer and in cancers affecting the stomach, intestine, and tongue.

**Malarial Cachexia.**—By this is meant the changes which are apt to supervene in an individual who has suffered from one or more attacks of malarial fever, or, but to a less extent, who has been for long times exposed to malarial poison. It is associated specially with changes in the blood and spleen, but the liver, kidney, and nervous system are apt also to show morbid changes. The cachexia shows itself principally in the colour of the skin. It is pale, resembling in this respect ordinary anaemia, but it often reveals a yellow aspect, which has been compared to that of old wax, or a brownish colour, which has been compared to that of gingerbread. With this the affected individual usually presents an aspect of marked feebleness, and shows a proneness to dropsy of the dependent parts.

**Metallic Cachexias.**—Of these the lead and the mercurial are the examples which have received most attention. The lead is the only one which merits here detailed reference. This cachexia, which rapidly manifests itself in individuals who have been exposed to the causes of lead poisoning, consists mainly in an alteration in the colour of the skin. This in its pallor resembles anaemia, but differs from it in presenting, along with the pallor, an appearance of what is best expressed by dirtiness, the result of the presence in the pale skin of minute particles of lead. In addition, the blue line along the softened gums, the factor of the breath, and the peculiar paralysis, etc., render its recognition easy.

**Cachexia Africana.**—African or Negro cachexia—the result, perhaps, of dirt-eating among the African natives, cachexia aquosa, possibly the same disease as miner's anaemia.

**Cachexia Saturnina.** See CACHEXIA (Metallia).

**Cachexia Strumipriva.**—A morbid state, allied to myxoedema, operative myxoedema. See THYROID GLAND, MEDICAL.

**Cacodyl.** See TOXICOLOGY (Arsenic)—Cacodyl, or diacodyl, or disentiaramethyl, exists as cacodyl oxide ( $As_2O_3(CH_3)_2$ ) or Cadet's fuming liquid, it has a powerfully offensive odour, from it cacodylic acid is obtained by exposure to the air, there are also salts, such as sodium and ferric cacodylate, which are used medicinally, a salt somewhat analogous to sodium cacodylate is employed in medicine as *antihemal*, and has tonic properties.

**Cacoethes.**—Cacoethes means literally a bad habit, or an ill-conditioned or even in dignant state. The inveterate tramp, well known to workhouse authorities, is said to have the *cacoethes ambulandi*.

**Cacogenesis.**—Abnormal formation, such as is seen in teratological or pathological conditions.

**Cacopathia.**—A severe malady of the mind (or body).

**Cacosmia.**—The perception of a bad odour, due either to its actual existence (foreign body in nose, disease of sinuses, etc.), or to a disorder of the olfactory nerve tract. See NOSE, NASAL NEUROSES.

**Cacothymia.**—A depressed mental condition, with deprivation of the morals.

**Cadaveric Rigidity.** See MEDICINE, FORENSIC (*Signs of Death, Rigor Mortis*), PREGNANCY, EXTRA-UTERINE DISEASES (*Death of Fetus, Rigor Mortis*).

**Cadaverine.**—A ptomaine ( $C_4H_{10}N_2$ ) obtained from human remains in the later stages of decomposition, a liquid having a very disagreeable smell. See IMMUNITY (*Immunitation with Toxin Filtrate*), SNAKE-BITES AND POISONOUS FISHES (*Putrefaction of Fish*), URINE, PATHOLOGICAL CHANGES IN (*Diaminuria*).

**Cade, Oil of.**—Juniper tar oil or *Oleum Cadumum* is a viscid, oily liquid, with a tar-like smell, prepared by distillation of the wood of the *Juniperus Oxycedrus*, and acting beneficially in such skin diseases as eczema, psoriasis, and pruritus. It is used as an ointment (e.g. melted with an equal part of yellow wax).

**Cadenabbia.** See THERAPEUTICS, HEALTH RESORTS (*Italian Lakes*)

**Cadmium.**—Cadmium (Cd) resembles zinc in its chemical relations, in the form of the sulphate it acts locally as an astringent, it is not official. The iodide (ten grains to one ounce of vaseline) has been used as an ointment in the erythematous stage of acne rosacea.

**Caduca.**—The decidua. See FETUS AND OVUM, DEVELOPMENT OF (*Decidua*)

**Caduca Passio** or **Caducus Morbus.**—Epilepsy, or the "falling sickness."

**Cæcitas.**—Blindness, e.g. *cæcitas verbalis*, word-blindness, *cæcitas nocturna*, hemeralopia.

**Cæcum.**—The caput coli of the intestine. See APPENDIX VERMIFORMIS, HERNIA (*Congenital Hernia of Cæcum*). TYPHITIS

**Cænesthesia.** See CYNÆSTHESIS

**Cæsalpinia.**—A genus of shrubs and trees of the Natural Order of the Leguminosæ. The seeds of *C. aculeata* are used in malaria, and those of *C. Bonduca* as a tonic and anthelmintic as well as in malaria, the leaves of the latter are said to act as an emmenagogue. The *C. pulcherrima* has seeds and leaves which are diuretic, purgative, and abortifacient, its pods are astringent. The seeds of the *C. Sappan* of India act as emmenagogues.

**Cæsarean Section.** See LABOUR, OPERATIONS, LABOUR, PROLONGED (*Contracted Pelvis*), PREGNANCY, AFFECTIONS OF GENITATIVE ORGANS (*Fibroid and Ovarian Tumours*)

**Cæsium.**—An element (Cs) having an atomic weight of 133, it resembles potassium, and is contained in the rare mineral *pollucite*. Bromide of cæsium has been used as a sedative in place of bromide of potassium.

**Caffeic Acid.**—Caffeic acid, *caffotannic acid*, and *caffic acid* are acids obtained from coffee, *caffone* is the aromatic principle of coffee, and *caffendine* is an alkaloid ( $C_7H_{12}N_4O$ ) got by decomposing caffeine (*q v*).

**Caffeina.** See also ALKALOIDS, COLOUR VISION (*Acquired Colour-blindness*), DIURETIC, GUARANA, PHARMACOLOGY, etc.—An alkaloid ( $C_8H_{10}N_4O_2 \cdot H_2O$ ) obtained from common tea (*Camellia thea*) or coffee (*Coffea arabica*), and known also as *Thème* or *Guaranine*. It is a methyl derivative of xanthine, and is thus related to theobromine, for caffeine is trimethyl-xanthine, while theobromine is dimethyl-xanthine. Caffeine can be made quite soluble in cold water if half a gram of sodium sacchylate for each gram of the caffeine be added. It may be given in doses of 1 to 5 grains, but its salt the citrate

is more often employed. *Caffeina Citras* is official, and may be given in doses of 2 to 10 grains, or in effervescent form (*Caffeina Citras Effervesce*) in doses of 60 to 120 grains. Incompatibles are potassium iodide, mercurial salts, and tannic acid.

The chief action of caffeine is to increase the force of the heart's action and to raise the blood-pressure. It also stimulates mental activity, and acts as a diuretic. It is used in heart disease for its stimulant action, and it is specially useful when there is concomitant dropsy. It has the additional advantage that it can, when mixed with solution of sodium sacchylate, be given hypodermically. It is used in poisoning by alcohol and opium. The *valerianate* has been used in whooping-cough and hysteria, and the *arsenate* in malaria.

**Cagot.**—Cagot is the name given to certain proscribed races living chiefly in the Basque Provinces. They were not cretins, possibly they suffered from a mild form of liposity. The "Cagot Ear" has been regarded as a characteristic malformation, but this seems to be open to question.

**Caisson Disease.** Compressed in Disease of Diver's Phlegm. See SPINE, SURGICAL AFFECTIONS OF, VERIGO.

**Cajuput Oil.**—The volatile oil (*Oilum Cajuputi*) is derived from the leaves of *Melaleuca leucadendron* (or *M. minor*), and is a bluish-green liquid containing hydrous cajuputene, cajuputol, or cineol (eucalyptol),  $C_{10}H_{18}OH$ , and terpineol, as well as aldehydes (butyric, benzoic). It is given in doses of  $\frac{1}{2}$  to 3 m, or as the *Spiritus Cajuputi* in doses of 5 to 20 m. Internally it is taken (on sugar) for colic, toothache, neuralgia, nervous vomiting, dysmenorrhœa, etc., acting then as a carminative and antispasmodic; externally, it is used as a stimulant and counter-irritant, as in chilblains and chronic rheumatism.

**Calabar Bean.** See PHYSOSTIGMATIS SEMINA, TOXICOLOGY (*Alkaloids*)

**Calamine.**—Native zinc carbonate, calamina preparata contains chiefly zinc carbonate with oxide of iron, etc.

**Calamus Scriptorius.**—The lower end of the fourth ventricle of the brain, terminating in a pen-shaped point. See PHYSIOLOGY (*Nervous System, Vaso-motor Mechanism*)

**Calcaneo-Cavus.**—A variety of club-foot (*q v*)

**Calcaneum.**—The os calcis or heel bone. See ANKLE-JOINT, INJURIES (*Fracture of Os calcis*)

**Calcareous Concretions and**

**Deposits.** See MENINGES OF THE CEREBRUM (*Minor Conditions*); TONSILS (*Chronic Tonsillar Abscess*); TUBERCULOSIS (*Morbid Anatomy*); TUMOURS (*Myomata*).

**Calcareous Degeneration.** See ARTERIES, DISEASES OF; CALCIFICATION; HEART; MYOCARDIUM AND ENDOCARDIUM (*Infiltrations*); PREGNANCY, DISEASES OF PLACENTA; PREGNANCY, INTRA-UTERINE DEATH OF FETUS; TERTII (*Diseases of the Pulp*).

**Calcarine Fissure.**—A cerebral fissure joining the parieto-occipital fissure, and helping to mark off the cuneus. See BRAIN, PHYSIOLOGY (*Anatomical*); PHYSIOLOGY, NERVOUS SYSTEM (*Cerebrum*).

**Calcification.**—An abnormal deposition of lime salts (carbonate and phosphate) in the tissues, especially in those which are useless, decaying, or dead (e.g. blood-clot, dead fetus (lithopedion), caseous masses, senile arteries, heart-valves, pleura, pericardium, splenic infarcts, etc.). The cause is uncertain, but neither an excess of lime salts in the blood nor their resorption from bones has been proved; it is probable that a depreciation of the nutrition of the tissues is a necessary antecedent to calcification. See ARTERIES, DISEASES; LIVER, DISEASES OF (*Calcareous Infiltration*); TUBERCULOSIS (*Morbid Anatomy*).

**Calcium and its Salts.** See HEMOPHILIA (*Treatment*); PANCREAS, DISEASES (*Chronic Pancreatitis, Treatment*); PHARMACOLOGY; PRESCRIBING; RHEUMATISM, RHEUMATOID ARTHRITIS (*Urine*); STOOLS (*Enteroliths*); URINE, PATHOLOGICAL CHANGES IN (*Metals, and Calcium oxalate*).

*Calcium Carbonate* is official in two forms—1. *Creta Preparata*, a dull-white powder, insoluble in water. *Dose*—10-60 grs. *Preparations*—(1) *Hydrargyrum cum Creta*, grey powder. *Dose*—1-5 grs. (2) *Mistura Cretæ*. *Dose*— $\frac{1}{2}$ -1 fl. oz. (3) *Pulvis Cretæ Aromaticus*. *Dose*—10-60 grs. (4) *Pulvis Cretæ Aromaticus c. Opio*. *Dose*—10-40 grs. 2. *Calcii Carbonas Precipitatus*, a white insoluble powder. *Dose*—10-60 grs.

*Calcium carbonate* is protective and mildly astringent, and prepared chalk is therefore useful as a dusting powder and for application to moist eczema. It is also an excellent tooth powder, either alone or made into a paste with antiseptics. Internally its action is limited to the alimentary canal, and it is given for diarrhoea, especially in children, in the form of *Mistura Cretæ* or *Pulv. Cretæ Aromat.* It is also useful in some cases of dyspepsia with hyperacidity.

*Calcii Chloridum* is in the form of white masses, very hygroscopic, and soluble in their own weight of water. *Dose*—5-15 grs. It is given where there is a tendency to hemorrhage, on account of its power of increasing the coagula-

bility of the blood. In hæmoptysis, hæmatemesis, aneurysm, and so on, where the blood condition is not the cause of the hemorrhage, it is useless; but in hæmophilia, jaundice, and other diseases predisposing to bleeding it is of great benefit. Operations for abdominal diseases associated with jaundice should always be preceded by a course of calcium chloride.

*Calcii Hydras*, slaked lime, is obtained from the interaction of water and lime (Calcium Oxide). It is a white alkaline powder, soluble 1 in 900 of water; 1 in 60 if sugar be added. *Preparations*—(1) *Liquor Calcis* (lime water). *Dose*—1-4 fl. oz. (2) *Liquor Calcis Saccharatus*. *Dose*—20-60 m. (3) *Linimentum Calcis*, consisting of equal parts of lime water and olive oil. Slaked lime mixed with caustic potash has been used for destroying warts. *Linimentum calcis* is recommended for burns. "Carron oil" consists of equal parts of lime water and linseed oil. Lime water is used to prevent the curdling of milk in the stomach, and is very efficacious in the vomiting of infants when that is due to rapid curdling of the milk. It may also help in checking a mild diarrhoea.

*Calcii Phosphas*, derived from bone ash, is a white insoluble powder. *Dose*—5-15 grs. *Preparation*—*Syrupus Calcii Lactophosphatis*. *Dose*— $\frac{1}{2}$ -1 fl. dr. It has been used in anemia, rickets, and various forms of malnutrition, on purely theoretical grounds; but very little of it can be absorbed, and it is more than doubtful if it does any good at all. If used it should be given in large doses.

*Calcii Hypophosphis* is soluble in water. *Dose*—3-10 grs. It has been used for the same conditions as the phosphate, and is probably more efficacious because of its solubility. The *Glycerophosphate of Calcium* is very popular as a tonic, and it certainly seems to do good in some cases.

*Calc. Sulphurata* is a dull-grey insoluble powder with an odour of sulphuretted hydrogen. *Dose*— $\frac{1}{2}$ -1 gr. It has been recommended for all kinds of suppurative processes, particularly intractable furunculosis. There is no proof that it has any effect whatever in checking or relieving such conditions.

**Calculus.** See BLADDER, INJURIES AND DISEASES OF (*Calculus Vesicæ*); CHILDREN, CLINICAL EXAMINATION OF (*Urinary System*); GALL-BLADDER AND BILE DUCTS, DISEASES OF (*Cholelithiasis*); KIDNEY, SURGICAL AFFECTIONS OF (*Pyelitis, Stone in the Kidney*); LACHRYMAL APPARATUS, DISEASES OF (*Excretory Apparatus, Calculi*); NOSE, FOREIGN BODIES (*Rhinoliths*); OBESITY (*Pathological Relations*); PANCREAS, DISEASES OF (*Pancreatic Lithiasis*); PENIS, SURGICAL AFFECTIONS OF (*Preputial Calculi*); PREGNANCY, PLACENTA, DISEASES OF (*Calcareous Degeneration*); PROSTATE GLAND (*Prostatic Concretions*); SCROTUM AND TESTICLE, DISEASES OF (*Scrotal Calculi*); URETHRA, DISEASES OF (*Cal-*



*culi*), URINE, PATHOLOGICAL CHANGES IN (*Urinary Calculi*), VESICULÆ SEMINAE (*Calculi*)

**Calcutta Fever.**—Bastard typhoid fever. See TROPICS, THE UNCLASSED FEVERS OF THE

**Caldarium.**—The hot chamber in the ancient Roman baths. See BALNEOLOGY (*Historical*)

**Caldas da Rainha.** See BALNEOLOGY (*Portugal*)

**Caldas-de-Gerez.** See BALNEOLOGY (*Portugal*)

**Caldas-de-Montbuy.** See BALNEOLOGY (*Spain*)

**Caledonia Springs.** See BALNEOLOGY (*Canada*)

**Calefacient.**—Causing heat, e.g. *Emplastum Calefaciens*. See CANTHARIDES

**Calendar, Obstetric.** See PREGNANCY, DIAGNOSIS (*Probable Date of Confinement*)

**Calenture.**—Fever, especially of thermic causation, affecting sailors more particularly, in the tropics, thermic fever or heat apoplexy, syniasis. See SUNSTROKE

**Calf-Lymph.** See VACCINATION

**Calgary.** See THERAPEUTICS, HEALTH RESORTS (*American, British Columbia*)

**Calico-Dyers.** See TRADES, DANGEROUS (*Lead-Poisoning*)

**California.** See THERAPEUTICS, HEALTH RESORTS (*American*)

**Calliper - Compasses or Callipers.**—A sort of compasses, with attached scale, for measuring the diameters of convex bodies, such as the fetal head (*Cephalometer*) or maternal pelvis (*Pelviometer*). See LABOUR, PRECIPITANT AND PROLONGED (*Pelviometry*)

**Callirrhoe.** See BALNEOLOGY (*Turkey, Palestine*)

**Callisthenics.**—Simple gymnastic exercises, employed to prevent or cure spinal curvature, especially in young girls. See SPINE, SURGICAL AFFECTIONS OF

**Callositas.**—A local thickening of the horny layers of the skin (*hyperkeratosis*), occurring especially on the palms and soles, due to continuous pressure or irritation from the handling of tools, etc. See LEUCLYOSIS (*Callositas*)

**Callus.**—Bony material thrown out between and around the ends of a fractured bone

during the healing process, especially marked if the fracture has been badly set. See FRACTURES (*Process of Repair*), LABOUR, PROLONGED (*Pelvic Deformities, Fracture*)

**Calomel.**—Mercurous chloride. See MERCURY. See also CHOLAGOGUES, ECLAMPSIA, PHARMACOLOGY, PRESCRIBING, SYMPTOMS, etc.

**Calor Mordax.**—The dry, burning, pungent heat of the skin, as noted by the observer's hand, in pneumonia, malaria, and scarlatina. See PNEUMONIA, CLINICAL (*Clinical Features*)

**Calorie or Calory.**—The conventional unit of quantity of heat: the small calorie is the amount of heat necessary to raise 1 gram of water 1° C° (or from 15° to 16° C°), the large calorie is that required to raise 1 kilogram of water 1° C°, and is equivalent to 3.97 British thermal units (the B.Th.U. = quantity of heat necessary to raise 1 lb. of water 1° F°). See PHYSIOLOGY, TISSUES (*Muscular*), PHYSIOLOGY, FOOD AND DIGESTION (*Energy Value*)

**Calorigen.**—A form of gas stove (George's), containing pipes conveying a supply of fresh air

**Calotropis.**—*Madu*, or the dried root of *Calotropis procera*, official in India and British Colonies, used in doses of 3 to 10 grs. as a tonic, and of 30 to 60 grs. as an emetic, the *Tinctura Calotropis* has a dose of  $\frac{1}{2}$  to 1 fl. dr., it is employed for the treatment of elephantiasis (externally), and of syphilis and rheumatism

**Calumbæ Radix.** See GENTIAN, PHARMACOLOGY, PRESCRIBING, QUASSIA, etc.—*Calumbæ Root* is a typical vegetable bitter. Its chief constituents are—(1) *Calumban*, a neutral bitter principle, (2) *Beberine*, an alkaloid, (3) *Calumbic Acid*, (4) *Starch*. The active principle may be looked upon as a mixture of the first three. It contains no tannin, and its preparations can therefore be prescribed with salts of iron.

*Preparations*.—1 Infusum Calumbæ. *Dose*.— $\frac{1}{2}$ -1 oz. 2 Liquor Calumbæ Concentratus. *Dose*.— $\frac{1}{2}$ -1 dr. 3 Tinctura Calumbæ. *Dose*.— $\frac{1}{2}$ -1 dr.

*Calumbæ* is administered before food as a stomachic tonic, either alone or, more usually, in combination with other drugs, such as iron, arsenic, and nuxvomica. It stimulates the appetite, and increases the flow of saliva and gastric juice. It is useful in cases of weak primary digestion, in general debility, in anemia, and during convalescence from acute diseases. The infusion has been given as a rectal injection to destroy thread-worms, but quassia is more generally employed for this purpose.

**Calvaria.**—The top part or roof of the skull, the portion lying above the occipital protuberance, the orbits, and the ears, *calvarium* is a modern form of the word. See ANTHROPOLOGY.

**Calvities.**—Baldness. See ALOPECIA.

**Calx.** See CALCIUM, CHLORINE (*Calc Chlorinata*), SULPHUR (*Calc Sulphurata*), PHARMACOLOGY, PRESCRIBING.

**Cambogia.**—Gamboge is a gum resin, acting as a powerful purgative (diastic hydragogue), and seldom used (on account of its griping effects), unless in the form of the *Pilula Cambogia Composita* (which contains also Barbados aloes). Gamboge contains a bright yellow resin (gambogic acid) and a soluble gum; the dose is  $\frac{1}{2}$  to 2 grs, and of the Compound Pill, 4 to 8 grs. *Cambogia Indica*, or Indian Gamboge, is found in the Indian and Colonial Addendum (1900) to the British Pharmacopoeia; it is got from *Garcinia morella* (the British drug is from *Garcinia Hanburii*), and it has the same action and dose ( $\frac{1}{2}$  to 2 grs).

**Cameron's Septic Tank.** See SEWAGE AND DRAINAGE (*Sewage Disposal*).

**Camisole.**—A strait-jacket or strait-waistcoat, used in the management of the insane.

**Camp Fever.** See TYPHUS FEVER.

**Camphene.**—One of the series of solid hydrocarbons, called *terpenes* ( $C_{10}H_{16}$ ), formed artificially or occurring naturally (in some oils).

**Camphora.** See APHRODISIACS, PHARMACOLOGY, PRESCRIBING, etc. — *Camphora* is obtained from *Cinnamomum camphora*, the camphor laurel, found in the East Indies, China, and Japan. It is in the form of colourless crystalline masses, with a powerful characteristic odour and a bitter, pungent taste. It is soluble 1 in 700 of water, 1 in 2 of oil of turpentine, 1 in 4 of olive oil, and readily in milk, ether, alcohol, and chloroform. When mixed with chloral, carbolic acid, or thymol it forms a thick liquid. Dose—2.5 grs.

**Preparations.**—1 Aqua Camphoræ. Dose—1-2 oz. 2 Linimentum Camphoræ (Camphorated Oil). 3 Linimentum Camphoræ Ammoniatum (Compound Liniment of Camphor). 4 Spiritus Camphoræ. Dose—5-20 m. 5 Tinctura Camphoræ Composita (see OPIUM).

Liniments containing camphor are largely employed in chronic rheumatism, neuralgia, lumbago, sciatica, and in slight chest troubles. The benefit derived is probably due in most cases to its slight counter-irritant effect, in some to its local anæsthetic action. A mixture of camphor and carbolic acid dropped into a tooth will frequently relieve toothache. Internally,

camphor is used as a carminative and antispasmodic, especially in neurotic subjects. A few drops of the spirit of camphor in milk or on sugar taken every half-hour will in some persons arrest a cold in the head if taken in the initial stages. It has been used in cholera because of its mild antiseptic action. Lastly, it has been recommended in threatened cardiac failure, given hypodermically dissolved in rectified spirit (1 in 5).

**Campylorrhachis.**—Distortion or curvature of the spine, especially when of a tortuous or anomalous form. The same root (*καμπύλος*, crooked) is contained in *campylocheilus* (distorted hard), *campylorhinus* (crooked nose), and *campylorachis* (crooked leg).

**Canada.** See BALNEOLOGY (*America and Canada*).

**Canal Boats.**—Any vessel, not a ship, used for the conveyance of goods along a canal ("any river, inland navigation, lake, or water, being within the body of a county, whether it is or is not within the ebb and flow of the tide"), however it may be propelled. All such boats must (in England and Wales) be registered with the Local Authority if used as a dwelling-house. Regulations for the hygiene of canal boats, and for the management of cases of illness occurring on board, are found in the Canal Boats Acts of 1877 and 1884.

**Canal, Cloquet's.**—The hyaloid canal in the vitreous humour, through which, in the antenatal state, the hyaloid artery passes to the lens.

**Canal, Genital.** See GENERATION, FEMALE ORGANS OF, LABOUR, PRECIPITATE AND PROLONGED, LABOUR, INJURIES TO THE GENERATIVE ORGANS, UTERUS, MALFORMATIONS OF THE, VAGINA, DISORDERS OF.

**Canal, Haversian.** See PHYSIOLOGY, TISSUES (*Bone*).

**Canaliculi.** See LACRYMAL APPARATUS, DISORDERS OF.

**Canalization.** (1) The conversion (during labour) of the utero-vagino-vulvar tract into a continuous canal of practically equal calibre for the transit of the infant. See LABOUR, STAGES AND DURATION. (2) The formation of channels, e.g. blood-vessels. And (3) a method of draining wounds.

**Canal of Nuck.** See HERNIA (*Oblique Inguinal*).

**Canals, Semicircular.** See PHYSIOLOGY, SENSES (*Internal Ear*), PHYSIOLOGY, NERVOUS SYSTEM (*Semicircular Canals*), VERTIGO (*Ménière's Disease*).

**Canary Islands.** See THERAPEUTICS, HEALTH RESORTS (*Islands of the South Atlantic*).

**Cancellous.**—Tissues (e.g. bone) having a spongy or lattice-like structure are called *cancellous*. See PHYSIOLOGY, TISSUES (Bone)

**Cancer.** See TUMOURS (*Epithelial Group*) See also ADRENALIN, APPENDIX VERMIFORMIS (*Cancerous Disease*), APPETITE (*Loss of*), BLADDER, INJURIES AND DISEASES (*Tumours*), BONE, DISEASES OF (*Tumours*), BRAIN, TUMOURS OF (*Carcinomata*), CACHEMIA (*Cancerous*), COLON, DISEASES OF (*Secundary Membranous Colitis*), GALL-BLADDER (*Tumours of*), GALL-BLADDER AND BILE DUCTS, DISEASES OF (*Tumours of the Bile Ducts*), HEREDITY, HIP-JOINT, DISEASES OF (*Tumours in Region of Hip*), INTESTINES, DISEASES OF (*Malignant Disease*), KIDNEY, SURGICAL AFFECTIONS OF (*Tumours*), LABOUR, PRECIPITATE AND PROLONGED (*Faults in the Soft Passages, Cancer of the Cervix*), LIVER (*Perihepatitis, Secundary*), MAMMARY GLAND, DISEASES OF (*Carcinomata*), MEDIASTINUM (*Mediastinal and other Intra-Thoracic Growths*), MICRO-ORGANISMS (*Cancer Bodies*), OESOPHAGUS (*Growths, Malignant*), ORBIT, DISEASES OF THE, PANCREAS, DISEASES OF (*Malignant Diseases*), PERITONEUM, TUMOURS OF (*Malignant*), PREGNANCY, AFFECTIONS OF GENITALIVE ORGANS (*Cancer of Uterus*), RADIUM, RECTUM, DISEASES OF THE (*Cancer*), STOMACH AND DUODENUM, DISEASES OF (*Cancer*), TUMOURS (*Chloroma*), TUMOURS (*Carcinoma*), UTERUS, MALIGNANT TUMOURS OF, VAGINA, DISORDERS OF (*Carcinoma*), X-RAYS (*Cancer and Sarcoma*)

**Canchasmus.**—Immoderate laughter, usually hysterical in nature. See HYSTERIA

**Cancroin.**—A toxin found in carcinomatous growths, and used simultaneously for the treatment of cancer (*Adankiewicz*)

**Cancrum Oris.** See STOMATITIS (*Gangrenous*) See also GANGRENE (*Intestine*), MRSELES (*Complications, Noma*), TYPHOID FEVER (*Complication*)

**Candela.**—A bougie, wax or medicated

**Candles.**—Feeble illuminating agents, made of tallow, stearin, paraffin, wax, spermaceti, and compositions, they have practically no bad effect on health, "one candle power" is the light given by a sperm candle burning at the rate of 120 grs per hour. See VENTILATION AND WARNING

**Cane Sugar.** See PHYSIOLOGY, FOOD AND DIGESTION (*Carbohydrates*)

**Canities.**—Greyness, premature or semile. See NAILS, AFFECTIONS OF (*Canities*), SKIN, PIGMENTARY AFFECTIONS OF (*Pigmentary Anomalies of Hair*)

**Cannabis Indica.** See BROMISM (*Treatment*), DRUG ERUPTIONS (*Cannabis Indica*), PHAR-

MACOLOGY, PRESCRIBING, TOXICOLOGY (*Indian Hemp*)—*Cannabis Indica* is derived from the dried flowering or fruiting tops of Indian hemp—*Cannabis sativa*. It contains an active principle, *Cannabinon*, two alkaloids, a glucoside, and a large amount of resin known as *chiruris*. In India the plant is eaten or smoked for its intoxicating effects. "*Huschsch*" is a confection of the drug, "*Bhang*" a drink made from the powdered tops. *Preparations*—1 *Extractum Cannabis Indicae* Dose— $\frac{1}{4}$  gr 2 *Tinctura Cannabis Indicae* Dose—5-15 in Is contained in *Tinctura Chloroformi et Morphiæ Composita*. The action of this drug is first of all stimulating to the central nervous system, causing an intoxication analogous to that produced by alcohol. Larger doses are followed by lassitude, muscular weakness, and eventually sleep. It does not cause constipation, nor does it diminish the secretion of urine, and for these reasons it has been recommended as an anodyne and hypnotic in diseases in which opium is unsuitable, such as locomotor ataxia, neuralgia, gastralgia, and epilepsy. The preparations of cannabis indica, however, are unreliable, and the action of the drug varies greatly in different individuals. In some even a moderate dose causes great cerebral excitement and violent waking delirium. It has now to a great extent dropped out of use. The tincture is difficult to prescribe elegantly, because of the presence of the resin which is precipitated by water.

**Cannea.** See BALNEOLOGY (*India, Ceylon*)

**Cannes.** See THERAPEUTICS, HEALTH RESORTS (*Riviera*)

**Cannibalism.** See ETHNOLOGY

**Cannula.**—A tube, usually containing a puncturing instrument ( trocar ), introduced into a cystic tumour or body cavity to allow the escape of fluid, it may also be used for the admission of air (as in tracheotomy), for curing stenosis of a canal, or for carrying a plug to the posterior nares (*Bellini's cannula*)

**Canor.**—A metallic tinkling sound heard on auscultation

**Canquoin's Paste.**—A paste consisting of zinc chloride, zinc oxide, and wheat flour, caustic in action

**Cantharides.** See DERMATITIS TRAUMATICA ET VENENATA, NEPHRITIS (*Etiology*), PHARMACOLOGY, PRESCRIBING, TOXICOLOGY (*Abortifacients*)—*Cantharis* or *Spanish Fly* is derived from the dried and powdered beetle, *Cantharis vesicatoria*, collected chiefly in Hungary and Russia. The powder is greyish brown with shining green particles in it, and has a strong, disagreeable odour. The chief constituents are *Cantharidin*, the active principle, and two oils,

one volatile, giving the smell, and the other green, yielding the colour

**Preparations**—1 Tinctura Cantharidis Dose —5-15 m, if frequently repeated, 2-5 m  
2 Acetum Cantharidis Strength—1 m 10  
3 Unguentum Cantharidis Strength—1 m 10  
4 Emplastrum Cantharidis Strength—1 m 3  
5 Emplastrum Calefaciens Strength—1 m 25  
6 Liquor Epispasticus Strength—1 m 2  
7 Colodium Venereum Strength—1 m 2

Cantharides is chiefly employed externally as a counter-irritant. It is very reliable, although somewhat slow in action. It is applied over all sorts of inflammatory and painful conditions. It should never be used in patients suffering from kidney disease, in debilitated persons, or children, as it may be absorbed by the skin and cause internal irritation, particularly of the kidneys. It should be applied over a small area at a time. When the plaster is used it should be removed as soon as the blister has risen, because of the risk of absorption. Cantharides is contained in many mixtures the object of which is to stimulate the growth of the hair. It is hardly ever given internally, but small doses of the tincture have proved beneficial in chronic gleet. It has a wide reputation among the laity as an aphrodisiac, but this action, being due solely to direct irritation of the genital organs, can only be accomplished by the administration of toxic doses.

**Canthoplasty.**—The reconstruction of the canthus of the eye or any plastic operation in its neighbourhood. See EYELIDS, AFFECTIONS OF (*Spasm of the Orbicularis Muscle*). **Canthorraphy** is the operation of suturing the canthus, while **canthotomy** is simple division of it. (The *canthus* is the angle, outer or inner where the upper and lower eyelids meet.)

**Caoutchouc.**—Indianrubber, or rubber, obtained by drying the milky juice of *Hevea brasiliensis* and other tropical plants. The official preparation is *Liquor Caoutchouc* (made by dissolving 1 oz of indianrubber in shreds in 10 fl oz of benzol and 10 fl oz of carbon bisulphide), used in the preparation of Charta Smapiis. Vulcanised indianrubber is a combination of sulphur with caoutchouc.

**Capacity.**—Testamentary capacity is the state of mental health necessary for making a will which shall be valid. See CIVIL INCAPACITY.

**Cape Town.** See THERAPEUTICS, HEALTH RESORTS (*South Africa*).

## Capillaries, Diseases of.

### CONGENITAL ANGIOMA—

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### NON-CONGENITAL CAPILLARY ANGIOMA

53

THE most common and important affection involving capillaries is angioma. This may occur in any part of the body, but is most frequent in the skin and subcutaneous texture, so that we may confine our description to that region. Probably the most elastic and comprehensive classification of the angiomas is the anatomical

{ Capillary = Telangiectasis, Erectile tumour, Cavernous tumour  
 Angioma { Venous = Phlebectasis  
               Arterial = Cirsoid aneurysm  
               Lymphatic = Lymphangioma

*Capillary angioma* is best considered under two heads—the congenital and the non-congenital.

**I CONGENITAL ANGIOMA NÆVUS**—may be subdivided pathologically or clinically. In the one view we have the simple, venous, and arterial, in the other the cutaneous, subcutaneous, and mixed. We shall presently see the value of these distinctions.

**Histology.**—On section the simple nævus presents the appearance of a finely reticulated sponge with imperfectly marked lobulation. The laminae are thin and fibrous, and lined by epithelium continuous with that of the blood-vessels. It may or may not be surrounded by a capsule. A nævus grows in two ways—*intimise* or *extimise*. If its growth is *intimise*, and especially if it be slow, it pushes parts aside and forms a capsule. But a nævus often invades its surroundings like an inflammation or a malignant tumour. What is the influence which metamorphosed capillaries can thus exercise on their neighbours it is difficult to conceive. But I have seen one grow by invasion in a week or two from the size of a shilling till it occupied half a baby's face and head, occluded its eye, and deformed its ear. In such cases, and in so far as a nævus may occupy the skin, there is no capsule, a point of importance in connection with treatment.

In the vascular arrangements there are many modifications. Thus it is not unusual for a nævus to pulsate from the beginning, when tortuous and dilated arterioles may be dissected out, although there is certainly no form which in the least resembles histologically the cirsoid aneurysm, or even perhaps the aneurysm by anastomosis of the adult. Similarly enlargement of venous radicles may enter largely into the composition of a nævus—so largely, indeed, as greatly to mask its original capillary character. If you put on the stretch skin which is occupied by a small and recent nævus, you will display a most beautiful network of visible capillaries often in parallel rows, with perhaps a winding arteriole or dilated vein. From this simple dilatation, and it may be multiplication of capillaries, presumably arise by pressure on the intervascular structures and formation of fresh intercommunications the appearances already described.

*Clinical History*—There may be seen a slight stain at birth, and as a rule a naevus declares itself, if at all, within a fortnight. It is very variable in its rate of growth. Its most rapid rate is generally early. After the first few months it tends to grow with the child. There is a great tendency to spontaneous disappearance. More than half of the subcutaneous and mixed varieties are naturally cured. The cutaneous variety, the port-wine stain, is, however, prone to persist through life. The subcutaneous and mixed varieties are likely to persist in proportion as they are associated with or acquire arterial, venous, or degenerative changes.

The natural cure is brought about by a fibrous atrophy probably inflammatory in character, and certainly capable of being excited by inflammatory causes to which a naevus is very sensitive. This process may be modified in various ways. In the atrophy portions of the tumour may be blocked off and form cysts. The altered blood in them is more thin and watery, and the deposit of fibrin on their walls less copious than in hæmatocele or extravasation cysts. I am not sure that these cysts are always blocked-off blood-vessels, because I have found them purely serous, even among others containing altered blood. But probably the most frequent degeneration is fibrous. The naevus is cured, but involution of the fibrous tissue fails. I have not met with a sarcomatous change in naevus unconnected with the pigmented mole.

*Diagnosis*—The symptoms need only be considered in so far as they aid diagnosis. The mixed variety of naevus may be mistaken for meningocoele and spina bifida, the subcutaneous or degenerated for fatty, cystic, and sarcomatous tumours.

The most important diagnostic difficulty is with meningocoele, which I have several times had sent me for naevus, from their similarity in appearance, consistence, and compressibility. The mere fact that the growth occurs in the middle line should put one on guard. In that line only lies the difficulty. The meningocoele is seen nowhere else. Cohesion to the bone calls for additional care. A naevus may cohere, but if on compression a neck-like connection, and still better if the contour of an office in the skull can be felt, a very important distinction is made out. If it be not absolutely congenital it is naevus. If an introduced needle be felt free in a cavity, and clear fluid with cerebral characters be removed, no doubt remains. The same considerations apply to spina bifida, which is also sometimes mistaken.

The sarcoma as it occurs in infants is apt to be very vascular, and looks like a naevus. The congenital, or nearly congenital origin, the shape, compressibility, consistence, and situation serve to distinguish a naevus from it and other tumours.

*Treatment*—No naevus in a child should be

submitted to treatment unless it be clearly increasing, or likely to do harm from its situation or condition. Many naevi disappear, and Nature's result is as good as any. The reason for treatment is not usually danger, but disfigurement. The danger comes from wound or ulceration. It is therefore slight. If the tumour be stationary, not ulcerated, and not doing harm, it is best to wait. I leave out of account harmless placebos, such as colloidion or gentle pressure.

The methods of treatment are very numerous.

They may be arranged as—

- 1 External escharotics or irritants, 2 Subcutaneous escharotics or irritants, 3 Ligature, 4 Excision.

1 There are three conditions under which this form of treatment should be adopted—

(a) A tiny naevus, with radiating vessels, is common on the nose or cheek. It generally goes away, but sometimes increases. It may be so easily destroyed by a needle with nitric acid, by a heated point, or the negative electrolysis needle, that it ought to be cured without delay.

(b) Flat naevi of the skin on the covered parts of the body may be well treated by Richardson's sodium ethylate. It leaves little scarring.

(c) Our only method of treating the port-wine stain of the face without leaving a worse mark is by the long-continued application of irritants. Squire's knife, multiple puncture, and all other means have with me been very disappointing. Cure by means of an irritative dermatitis is not certain, and requires much patience and perseverance, but it does no harm, and is sometimes successful. I think strong iodine is the most manageable irritant.

2 I have entirely given up the subcutaneous injection of such substances as tannin, perchloride of iron, and carbolic acid, as well as the subcutaneous use of the heated wire, in favour of electrolysis. All of them act in the same way, viz by the destruction of texture. But by electrolysis the destroying agents are introduced in infinitesimal division, and therefore are much more under the surgeon's control than by any other method. Moreover, the danger of subcutaneous injection without a temporary ligature has been frequently demonstrated by the sudden death of the patient. There are one or two rare cases in which the platinum wire heated by electricity subcutaneously might be used, but almost invariably for subcutaneous use electrolysis is the best agent.

The mode of using it and its value will be considered under the article "Electrolysis." Meantime it suffices to say that for mixed and subcutaneous naevi on exposed parts of the body, and for naevi elsewhere, which are very large or otherwise inoperable, electrolysis is safe and certain, although slow.

**Ligature**.—I think there is scarcely any case in which this method is now required.

**Excision**.—The advantages of excision over electrolysis are its greater certainty and rapidity. The former is slight, because excision may fail, and electrolysis rarely does so. The latter is undoubted and considerable. Excision requires only one operation, electrolysis usually more. In cases, therefore, where a scar is of no importance, and the size and relations of the tumour are not such as to make the operation dangerous, excision is preferable to any other method.

II. NON-CONGENITAL CAPILLARY ANGIOMA is in the simple form rare, most usual in internal organs, of exceedingly slow growth, but with little tendency to retrogression. More common are the venous and arterial. These three varieties correspond to the varieties of nevus histologically. They present, as above seen, differences from nevus clinically, and to the last belongs that rare affection called osteo-aneurysm. A fourth variety has no analogy among congenital varieties, and to it the name aneurysm by anastomosis may properly be given. It consists of metamorphosed capillaries in which large cavities are connected with dilated arteries on the one hand and dilated veins on the other. Many of the characters of arterio-venous aneurysm are here to be seen mingled with others more proper to a capillary or venous tumour.

In treatment we are here also restricted to electrolysis and excision. The former most certainly should be tried first, as it is practically without risk, and holds out a fair prospect of success. These varieties, and especially the aneurysm by anastomosis, are, however, much more resistant than congenital forms. When electrolysis fails the propriety of attempting excision must be determined in each case by the risk. It is always to be remembered that these tumours have little tendency to shorten life, and that only the trouble they give, chiefly by disfigurement, pulsation, and murmur, warrants interference. Operation by excision is always serious, often impossible.

The consideration of the other forms of angioma limited in origin to the arteries, to the veins, and to the lymphatics, will be found in the articles on these subjects and on "Tumours."

**Capillary Bronchitis.** See BRONCHITIS, BRONCHITIS (*Children*)

**Capital, Life.** See LIFE CAPITAL

**Capots.**—Cietins, probably a corruption of CAGOTS (*qv*)

**Capri.** See THERAPEUTICS, HEALTH RESORTS (*Italy, South*)

**Capric Acid.**—A monobasic, fatty acid ( $C_{10}H_{19}O_2$ ) occurring in goat's milk, it belongs to the acetic series (formic, acetic, propionic, etc.). *Caproic acid* ( $C_6H_{12}O_2$ ) and *Caprylic acid* ( $C_8H_{16}O_2$ ) are other acids of the same series. Each has its corresponding aldehyde ( $C_{10}H_{19}O$ ,  $C_6H_{12}O$ ,  $C_8H_{16}O$ ) and ether. *Caprin*, *caprom*, and *caprylin* are fatty substances found in goat's butter.

• **Capsici Fructus.**—The fruit (dried) of *Capicum minimum*, pod pepper. When powdered it is known as red pepper. The dose is  $\frac{1}{2}$  to 1 gr. There are two official preparations, the *Tinctura Capsici* (dose, 5 to 15 m), and the *Unguentum Capsici* (which resembles Smeiley's chille paste). It contains (along with other things) an acid substance (capsaicin), a volatile alkaloid (capsene), a volatile oil, and a resin. Externally, it is used (in the form of the ointment) as a counter-irritant in inflammatory and painful affections (pleurisy, sciatica), while internally it acts as a stomachic.

**Capsule, Internal.** See BRAIN, PHYSIOLOGY OF, PHYSIOLOGY, NERVOUS SYSTEM (*Cerebrum*)

**Capsules.**—Small gelatine cases, containing drugs (usually nauseous), for convenience in swallowing.

**Capsulitis.** See LIVER, PERIHEPATITIS, CATARACT, LENS, CRYSTALLINE

**Capsulotomy.**—(1) Incision of the capsule of the kidney as performed for Bright's disease (*Vide* DISCAPSULATION). (2) Incision of the capsule of the crystalline lens.

**Caput.**—The head (or origin) of a muscle, or of a bone, or of a part of the intestine ("caput coli"), etc. (*Vide infra*)

**Caput Medusæ.**—In the new-born infant the circum-umbilical cutaneous veins are apt to be dilated and tortuous, on account of stasis in the portal vein, the appearance thus produced is called the *Caput Medusæ*. See LIVER, DISEASES OF (*Morbid Anatomy, Anastomoses*)

**Caput Succedaneum.**—The soft swelling (serous infiltration) which forms over the presenting part (vertex, face, breech, shoulder) of the infant in labour. See LABOUR, STAGES AND DURATION (*Second Stage, Phenomena*), LABOUR, DIAGNOSIS AND MECHANISM (*Vertex, Face, Breech, and Breech Presentations*), LABOUR, PRECIPITATE AND PROLONGED (*Contracted Pelvis*)

**Carangidæ.**—Horse mackerel, poisonous fish. See SNAKE-BITES AND POISONOUS FISHES

**Caratés.**—A South American skin disease,

characterised by the occurrence of coloured patches on the face (the name signifies "look at the face"). See PINTA

**Caraway Fruit.** See CARUI FRUCTUS

**Carbalite.** - A mixture containing charcoal, used as a filter for water

**Carbamide.** - Urea ( $\text{NH}_2\text{CO NH}_2$ ), the diamide of carbonic acid, the first organic compound prepared by synthesis from inorganic sources. *Carbami* acid is carbamide with hydroxyl in place of amidogen ( $\text{NH}_2\text{CO OH}$ ), and it forms *carbamates* (e.g. ammonium carbamate, and ethyl carbamate or *urethane*)

**Carbazotic Acid.** - Picric acid or trimetaphenol

**Carbide.** - A compound formed by the union of carbon with another element, usually a metal, e.g. calcium carbide ( $\text{CaC}_2$ ).

**Carbo.** See also BISULPHIDE OF CARBON. SKIN, PIGMENTARY AFFECTIONS (*Hair*) - *Carbo Ligni*, wood charcoal, is the only official form of carbon. It is a black, odourless, and tasteless powder. *Dose* - 60 to 120 grs

Externally, charcoal has a considerable reputation as an application for foul ulcers and sores. It should be used in the dry state, and frequently renewed, as the discharge soon moistens it and destroys its oxidising powers. It is sometimes employed as a tooth powder. Internally, it has been given as a powder, as lozenges, and as biscuits to check gastric fermentation. It appears to have a special attraction for alkaloids, and has been recommended in large doses as an antidote in opium poisoning

**Carbohydrates.** See DIABETES MELLITUS (*Physiological Considerations*), DIET (*Nutritive Constituents of Food*), DIGESTION AND METABOLISM (*Carbohydrates*), ENZYMES (*Hydrolysis*), OBESITY (*Dietetic Treatment*), PHYSIOLOGY, FOOD AND DIGESTION (*Food, Heat Production, General Metabolism, and Diabetics*)

**Carbohydraturia.** - The presence of carbohydrates (sugars) in the urine

**Carbolic Acid.** See also ASEPTIC TREATMENT, ANTISEPTIC SURGERY, DRUG ERUPTIONS (*Tar*), GANGRENE (*Varieties, Carbolic Acid*), PHARMACOLOGY, PRESCRIBING, TETANUS (*Buccell's Method of Treatment*), TOXICOLOGY (*Corrosives, Acids*) - *Carbolic Acid*, or *Phenol*, is made from coal tar by distillation and purification. It consists of colourless acicular crystals which are very hygroscopic, and become liquid on the addition of 6 per cent of water. It melts into an oily liquid at  $91.5^\circ\text{F}$ . It is soluble 1 in 14 of water, and freely in alcohol and oils. *Dose* - 1-3 grs. - *Preparations* - 1. Acidum Carboliceum Laque-

factum. Contains 10 per cent of water. *Dose* - 1-3 m. 2. Unguentum Acidi Carbolici. 3. Glycerinum Acidi Carbolici, 1 gr of phenol in 5 m. 4. Suppositoria Acidi Carbolici, 1 gr in each. 5. Trochiscus Acidi Carbolici, 1 gr in each. Crude carbolic acid is used very largely as a disinfectant and deodorant for drains, bed-pans, and so on. The purified form in various dilutions is the most generally reliable antiseptic we possess, and is employed almost universally for surgical purposes. A 1 in 20 solution is used for purifying the surgeon's hands, the skin of the patient, etc. Instruments and dressings may be kept in a 1 in 20 or weaker solution till required. Dressings for wounds are soaked in a solution of 1 in 40 or 1 in 60, but care must be taken that too large a dressing is not applied, on account of the risk of absorption which takes place very readily through the skin. Fingers and toes should never be treated by carbolic fomentations, as numerous cases of gangrene following its use have been reported. Pure carbolic acid is a very efficient antiseptic for application to foul sores, ulcerated surfaces, abscess cavities, tuberculous sinuses, etc. A vaginal douche of 1 in 40 or 1 in 60 carbolic is very serviceable in the treatment of puerperal septicæmic conditions. Itchiness of the skin in scarlet fever is relieved by sponging with a 2 per cent lotion of carbolic. The ointment or a lotion is recommended for various skin diseases when a combined antiseptic and anæsthetic action are required. Glycerinum Acidi Carbolici is very efficient in destroying the fungus of tinea tonsurans. A small piece of cotton-wool soaked in pure carbolic, placed in a carious tooth, relieves toothache. The glycerinum is a useful application for aphthous or ulcerative stomatitis. It must be used carefully, as it is very powerful. It is also beneficial in septic conditions of the throat or tonsils, for which purposes it may be applied directly undiluted, or employed as a gargle or spray in the strength of 20 m of the glycerinum to the ounce of water.

Internally, carbolic acid has been advocated for dyspepsia with flatulence, diarrhoea, typhoid fever, and cholera, but its value in these diseases is very doubtful, on account of the dilution effected by the gastric and intestinal contents. Large doses of a 2 per cent solution administered hypodermically have given good results in some cases of tetanus. Its use in phthisis, internally, by inhalation, and as an intratracheal injection, has been abandoned. It is strongly advocated, however, as an inhalation in whooping-cough.

*Solus Sulphocarbolus* has been recommended to control gastric fermentation in doses of from 5 to 15 grs, but it has probably no beneficial action.

*Zinci Sulphocarbolus* may be used externally as an antiseptic for the same purposes as carbolic acid, but it has no special advantages.

**Carboluria.** See CARBOLIC ACID, URINE, PATHOLOGICAL CHANGES IN (*Colour, Drugs in*)

**Carbon Bisulphide.** See BISULPHIDE OF CARBON. See also AMBLYOPIA (*True*), TOXICOLOGY (*Organic Poisons, Carbon Bisulphide*), TRADES, DANGEROUS (*Bisulphide of Carbon*)

**Carbonic Acid.** See AIR, EXAMINATION OF (*Carbonic Acid, Ground Air*), BREATH (*Chemical Examination*), CHOLERA, EPIDEMIC (*Analysis of Symptoms, Respiration*), LABOUR, PHYSIOLOGY OF (*Causes of Labour*), MEDICINE, FORENSIC (*Death from Asphyxia*), MINERAL WATERS (*Muriated Saline, Earthy and Calcareous*), PHYSIOLOGY, BLOOD (*Gases*), PHYSIOLOGY, EXCRETION (*Urine*), PREGNANCY, PHYSIOLOGY (*Changes in Respiration*), STOMACH AND DUODENUM, DISEASES (*General Symptomatology, Flatulence*), TOXICOLOGY (*Gaseous Poisons, Carbon Dioxide*)

**Carbonic Oxide.** See ASPHYXIA (*Causes*), MEDICINE, FORENSIC (*Death from Asphyxia, Carbonic Oxide*), PHYSIOLOGY, BLOOD (*Carbon-monoxide-haemoglobin*), TOXICOLOGY (*Gaseous Poisons, Carbon Monoxide*)

**Carbonyl Chloride.**—An irritating gas ( $\text{CO}_2\text{Cl}_2$ ) formed by the decomposition of chloroform in the presence of burning coal gas or oil, phosgen. See ANAESTHESIA, GENERAL. PHYSIOLOGY (*Chloroform*)

**Carbuncle.** See ANTHRAX, BOILS AND CARBUNCLE (*Carbuncle*), DIABETES, MELITUS (*Complications, Skin*), NECK, REGION OF (*Inflammatory Affections*), SKIN, BACTERIOLOGY OF (*Bacteria identified with Carbuncle*), SUPPURATION (*Treatment, Operative*) TYPHOID FEVER (*Complications, Cutaneous System*)

**Carburetted Hydrogen.**—Marsh gas, methane, or methyl hydride ( $\text{CH}_4$ ) is light carburetted hydrogen, and has, in rare instances, been expelled from the mouth in flatulent dyspepsia (*rude Indigestion, Flatulent Dyspepsia*), methane mixed with air is the fire damp of coal-mines, and may cause explosions. Ethylene or olefiant gas ( $\text{C}_2\text{H}_4$ ) is heavy carburetted hydrogen.

**Carcinoma.** See TUMOURS (*Epithelial Group, Carcinoma*). See also CANCER (for cross-references)

**Carcinosis.**—Cancer or the cancerous cachexia, the *canceroses* have been regarded as including tuberculosis as well as cancers.

**Cardamomi Semina.**—Cardamom seeds are got from the *Elettaria cardamomum*, they contain a volatile and a fixed oil, and have a pleasant taste, and they are used as a carminative and stomachic medicine. The official preparation is the *Tinctura Cardamoni Com-*

*posita* (dose,  $\frac{1}{2}$  to 1 fl dr), but cardamom is also contained in the *Pulvis Cretæ Aromaticus*, the *Tinctura Rhei Composita*, etc.

**Carden's Amputation.** See AMPUTATIONS (*Carden's*)

**Cardia.**—The heart, or, more commonly, the point where the œsophagus opens into the stomach. See ANGINA PECTORIS, etc.

**Cardiac.**—Belonging to the heart or to the œsophageal opening into the stomach. For *cardiac asthma*, see ASTHMA, for *cardiac contractions*, see HEART, PHYSIOLOGY OF, and PHYSIOLOGY (*Circulation*), for *cardiac crises*, see TABLE DORSALIS (*Symptomatology, Cranial Nerves*), for *cardiac diseases*, see HEART, MYOCARDIUM AND ENDOCARDIUM, HEART, NEURONES, HEART, CONGENITAL MALFORMATIONS for *cardiac pain*, see ANGINA PECTORIS, and for *cardiac sounds (fetal)*, see PREGNANCY, DIAGNOSIS.

**Cardialgia.**—Gastric pain, especially pyrosis or heartburn. See CHEST-WALL, AFFECTIONS OF (*Pain in the Chest*), INDIGESTION (*Symptoms*), STOMACH AND DUODENUM, DISEASES OF (*General Symptomatology, Eruptions*)

**Cardiomele.**—Hernia of the heart through the thoracic wall or into the abdomen.

**Cardiocentesis.**—Cardiocentesis or puncture of the heart as a means of treatment in cases of asphyxia, chloroform-narcosis, etc., is not free from danger, but its trial has been to some extent warranted by experimental evidence.

**Cardiodynia.**—Pain in or near the heart, *angina pectoris (q v)*

**Cardiogram.**—A tracing of the movement of the apex of the heart, obtained by the use of the *cardiograph*, which consists, in its simplest form, of a receiving and of a recording tambour connected by a tube. See PHYSIOLOGY, CIRCULATION (*Circulation through the Heart*), PULSE (*Heart Movements*)

**Cardiolith.**—A calcareous concretion occurring in the heart, *eq* on one of the valves.

**Cardiolysis.**—Resection of the ribs in cases of adhesion of the pericardium and great vessels to the sternum, lungs, diaphragm, and structures in the anterior and posterior mediastinum, for the purpose of relieving the heart's action (*Brauer*)

**Cardiomalacia.**—Simple softening of the heart, as in obstruction of a small branch of a coronary vessel. See HEART, MYOCARDIUM AND ENDOCARDIUM (*Morbid Processes, Affections of the Myocardium*), NEPHRITIS (*Different Clinical Types, Circulatory System in*)



**Cardiometer.**—An apparatus (*eg* Roy's) used to measure the output of the heart in experimental work. See *PHYSIOLOGY, CIRCULATION (Physiology of the Heart)*

**Cardioptosis.**—Displacement of the heart in a downward direction, occurring in association with enteroptosis or from accommodation. A *voluntary* form has been reported in which a man had such command over his diaphragm that he could dislocate his heart, stomach, and kidneys at will (*Abrams*)

**Cardiorrhexis.**—Rupture of the heart

**Cardo.**—The axis vertebra, literally a hinge

**"Carferal."**—A water-filter consisting of iron, charcoal, and clay

**Caries.**—Ulceration, generally applied only to that process as it affects a bone. See *CHILDREN, CLINICAL EXAMINATION OF (Neck, Stiffness), JOINTS, DISEASES OF (Definition of Terms), JOINTS, DISEASES OF (Tuberculous Disease), NOSE, ACCESSORY SINUSES, INFLAMMATION (Causes), SPINE, SURGICAL AFFECTIONS (Spinal Caries), TEETH (Dental Caries)*

**Caries Sicca.**—A chronic form of tuberculous arthritis, occurring chiefly in the shoulder and hip joints of adults. See *JOINTS, DISEASES OF (Tubercular Diseases, Types)*

**Carinated.**—Keeled or furnished with a central groove or ridge; the abdomen may be carinated in tuberculous meningitis in children. See *MENINGITIS, TUBERCULOUS AND POSTERIOR BASIC*

**Carisbad.** See *MINERAL WATERS (Thermal, Alkaline)*

**Carminatives.**—Medicines, such as aromatic or bitter substances, stomachics generally, ethers, camphors, and volatile oils, which increase the gastric and intestinal muscular action, and so tend to expel flatus. One of their uses is to prevent the griping caused by purgatives, and they are usefully combined with antacids in cases of acidity (*eg* with bicarbonate of soda, etc.). The *Carminative Tincture* of the British Pharmacopœia Conference contains (aidamons, strong tincture of ginger, oil of cinnamon, oil of caraway, oil of cloves, and rectified spirit, its dose is 2 to 10 in

**Carniferrin.**—A preparation containing meat and iron. See *CHLOROSIS (Treatment)*

**Carniferrol.**—A preparation containing peptonised meat and iron

**Carnification.**—The transformation of a tissue or organ into a substance of flesh-like

consistence and appearance, *eg* the lungs in atelectasis and inflammation

**Carnin.**—One of the so-called purin bases, an end product of the breaking up of the nucleus of muscle, its formula is  $C_7H_5N_4O_4$

**Carnivora.** See *ANTHRAX*

**Carnochan's Operation.**—The antial method of reaching and removing Meckel's ganglion in trigeminal neuralgia. See *NERVES, NEURALGIA (Trigeminal, Surgical Treatment)*

**Carotid Artery.** See *ANEURYSM (Neck), NERVES, NEURALGIA (Trigeminal, Ligature of Carotid), OESOPHAGUS (Foreign Bodies, Ulceration into Carotids), ORBIT, DISEASES (Pulsating Eryththalmus)*

**Carotin.** See *CAROTIN*

**Carpal Bones.** See *WRIST-JOINT, INJURIES (Fracture)*

**Carphology.**—Seeking or picking movements of the hands noticed in delirious patients, "picking the bedclothes." It is derived from *káphos*, chaff, and *léγω*, I collect, and it is a serious symptom in the delirium of typhoid fever

**Carpo-pedal Spasm.** See *TRIANY (Causation, Rickets)*

**Carpus.** See *WRIST-JOINT, INJURIES, WRIST-JOINT, DISEASES*

**Carrageen.**—Irish moss, the thallus of *Chondrus crispus*. See *INVALID FEEDING (Carrageen Jelly)*

**Carrastraca.** See *BALNEOLOGY (Spain and Portugal)*

**Carreau.**—The name given by French writers to cases of tubes mesenterica in which there is hardness and enlargement of the abdomen, the name signifies a tile or brick floor (See *MESENTERIC GLANDS*)

**Carrion's Disease.** See *VERRUGA PERUANA (History)*

**Carron Oil.**—Equal parts of lime water and linseed oil. See *BURNS AND SCALDS (Treatment), CALCIUM, etc*

**Carrotin.**—A colouring matter obtained from dried and powdered carrots, it is said to be used to give a colour to butter, its formula is regarded as  $C_{18}H_{25}O$ . See *PIGMENTS OF THE BODY AND EXCRETA (Lipochromes)*

**Carrots.** See *PHYSIOLOGY, FOOD AND DIGESTION (Vegetable Food-Staffs)*

**Cartilage.** See *PHYSIOLOGY (Tissues), JOINTS, DISEASES (Ulceration of Cartilage),*

**KNEE-JOINT, INJURIES** (*Dislocation of Semilunar Cartilages*), etc.—There are many cartilages bearing special names, *e.g.* Meckel's cartilage, ensiform cartilage, Santorini's cartilage, etc.

**Carul Fructus.** See PHARMACOLOGY, PRESCRIBING.—Caraway fruit, the aromatic carpels of an umbelliferous plant (*Carum carui*). There is an *Aqua Carui*, given in doses of 1 to 2 fl oz. The volatile oil, *Oleum Carui*, is the chief constituent, and it contains carvone ( $C_{10}H_{16}O$ ), camphene, carvol, and limonene, it is given in doses of  $\frac{1}{2}$  to 3 m. On account of their pleasant, spicy taste and aromatic odour, caraway carpels are used in making of powders, tinctures, etc. The oil is a carminative.

**Caruncle.**—A small fleshy outgrowth, existing normally in the body (*e.g.* the navel, the prostatic lobe, and the lacrymal caruncle at the inner canthus), or developed as a pathological production (*e.g.* the urethral caruncle, nemoma, or vascular tumour growing near the meatus urinarius in the female).

**Carunculæ Myrtiformes.**—The fleshy bodies which represent the hymen after that membrane has been torn in coitus and compressed during labour. See A L I A, DISEASES or (*Morbid Conditions of the Hymen*).

**Carus.**—Deep sleep, or, according to some, coma or sopor, *carus cataleptus* is simply catalepsy, *carus lethargus* is prolonged semi-consciousness or trance, and *carus ecstasis* is ecstasy. See CATAPLEXY, ECSTASY, INSANITY.

**Carvol or Carvone.**—A constituent ( $C_{10}H_{16}O$ ) of oil of caraway and oil of spearmint, *carvicol* is an isomeric form of it, and *carvone* ( $C_{10}H_{16}O$ ) is a terpene or limonene. See CARUI FRUCTUS.

**Caryocinesis.**—The changes in the nucleus occurring during mitosis or indirect cellular division. See PHYSIOLOGY, THE CELL.

**Caryophyllum.**—The dried flower-buds of *Eugenia caryophyllata*, or cloves, having a pungent and aromatic taste and a spicy odour, they contain caryophyllin, eugenin, and an oil (*Oleum Caryophylli*, or oil of cloves) which is official (dose,  $\frac{1}{2}$  to 3 m.). of cloves itself there is an official preparation, the infusion (*Infusum Caryophylli*), given in doses of  $\frac{1}{2}$  to 1 fl oz. Applied externally, oil of cloves acts as an irritant, and later as a local anæsthetic; internally, it is a stomachic and carminative, and by its action on the stomach it reflexly stimulates the heart and circulation, it is also used in toothache from decayed teeth, as a topical application. To prevent griping, oil of cloves is used in compounding the *Pilula Colocynthis Composita* and the *Pilula Colocynthis et Hyocyami*. See PHARMACOLOGY, PRESCRIBING, and VOLATILE OILS.

**Caryoplasm.**—The substance of the nucleus as distinguished from that of the cell (or cytoplasm), the endoplasm, the term has been somewhat indefinitely used for either the chromatin or the achromatin of the nucleus.

**Casamicciola.** See BATHOLOGY (*Italy, Ischia*).

**Cascara Sagrada.** See PHARMACOLOGY, PRESCRIBING, PURGATIVES.—The bark of the Californian buckthorn (*Rhamnus purshiana*), having a markedly bitter taste, and containing cascarn, purshianin, a volatile oil, resins, etc. There are three official preparations, the *Extractum Cascare Sagrade* (dose, 2 to 8 grs.), the *Extractum Cascare Sagrade Liquidum* (dose,  $\frac{1}{2}$  to 1 fl dr.), and the *Syrupus Cascare Aromaticus* (dose,  $\frac{1}{2}$  to 2 fl dr.). The *liquid extract* may be usefully combined with Spiritus Animonie Aromaticus, Spiritus Chlorotormi, Tinctura Belladonnæ, and Tinctura Nucis Vomice (equal parts of each), to form the *Tinctura Laxativa* (dose, 20 to 60 m.). As an aperient, cascara finds its great sphere of usefulness in the treatment of chronic constipation, it does not gripe much, it can be used daily without increasing the dose, and after the constipation has been relieved the drug can be gradually discontinued. It may be given in association with eucalyptin and nadin, with nuxvomica and belladonna, and with iron.

**Cascarillæ Cortex.**—The bark of *Croton Eluteria*, containing the bitter substance cascarnin, volatile oils, resins, etc. It has two official preparations, the *Infusum Cascarille* (dose,  $\frac{1}{2}$  to 1 fl oz.) and *Tinctura Cascarille* (dose,  $\frac{1}{2}$  to 1 fl dr.), and it acts as a bitter and stomachic medicine. There is an incompatibility between the tincture and mineral acids.

**Caseation.** See BRONCHITIS, BRONCHIAL GLANDS (*Morbid Anatomy*), TUBERCULOSIS.—A form of necrosis of the tissues in which they become converted into a cheese-like substance, it is seen typically in tuberculous, and it may follow either coagulation-necrosis or a granular disintegration of the tissues. It is more correct to term caseation a post-necrotic state than a form of necrosis (*Greenfield and Lyon*).

**Casein.**—A proteid substance occurring in milk (in the form of caseinogen, a nucleoproteid), and forming a great part of cheese. See PHYSIOLOGY, FOOD AND DIGESTION, MILK (*Physiological*), INVALID FEEDING, etc. Casein biscuits are used in diabetes mellitus.

**Cassia Pulpa.**—The pulp from the pods of the purging cassia (*Cassia fistula*), containing a purgative principle (cassim). It is contained in the official *Confectio Senna*, and is not given in any other way. See PHARMACOLOGY, PRESCRIBING, PURGATIVES.

**Castellamare.** See THERAPEUTICS, HEALTH RESORTS (*Italy*)

**Castellamari di Stabia.** See BALNEOLOGY (*Italy, Bay of Naples*)

**Castor.**—The dried preputial follicles (with their secretion) of the beaver (*Castor Fiber*), it contains a resinous matter, *castoreum* it is known officially as *Moschus* or *Musk* (*qv*)

**Castor Oil.** See CONSTIPATION, PHARMACOLOGY, PRESCRIBING, PURGATIVES, etc — *Oleum Ricini*, the oil expressed from the seeds of *Ricinus communis*, consisting chiefly of glyceryl ricinoleate,  $C_{18}H_{33}(C_{15}H_{27}O)_2$ , or *ricinoleine*, a fixed oil soluble in alcohol, *ricinine*, which has been described as an alkaloid with a formula of  $C_{21}H_{32}N_2O_8$ , is doubtfully so, and has no purgative properties, another constituent is *ricin*, an albumose resembling in its action the abrin of joriquity seeds. The dose of castor oil is 1 to 8 fl dr. It has a nasty taste and smell, only partly concealed in the official *Mistura Olei Ricini* (dose, 1 to 2 fl oz). Indeed, no plan of getting over the difficulty of the taste succeeds so well as the holding of the nose till the drug has been washed over the fauces by such a liquid as coffee, for if the nose be held only during the swallowing of the oil the smell is apt to pass up the posterior nares and cause nausea. Castor oil is a very valuable simple purgative, and is specially useful in children, in pregnant and puerperal women, and in adults and old people of both sexes who may be suffering from hemorrhoids. It may be given, with olive oil, as an enema. Externally, it is used sometimes as a sedative in cases of conjunctivitis (*eq* due to the introduction of an irritant into the eye), when it can be dropped into the eye, either alone or in combination with cocaine

**Castration.** See OBESITY (*Sex*), PROSTATE GLAND, HYPERPLASIA (*Treatment, Radical*), SCROTUM AND TESTICLE, DISEASES OF (*Excision of the Testicle*)

**Casts.** See BRONCHI, BRONCHITIS (*Morbid Anatomy*), HEMATURIA (*Blood Casts*), NEPHRITIS (*Clinical Features, The Urine*), URINE, PATHOLOGICAL CHANGES IN (*Casts, hyaline, amyloid, epithelial, blood, leucocyte, granular, fatty, and mastic*)

**Cata-**—In compound words "cata-" (or "kata-") has generally the sense of "down," "downward," "descending," "declining," but sometimes it means "perverted" or "thorough." It is contained in many words (as well as in those specially named below), such as *catacasmus* (deep scarification), *catacausis* (deep burning, or, perhaps, "spontaneous combustion"), *catachysis* (a douche), *cataclysm* (a clyster), *cataleptics*

(reflecting and refracting), *cataplasm* (a plaster), *cataptosis* (an apoplectic or epileptic fit), etc

**Catabolism.**—Catabolism (or katabolism) means the descending, disruptive, or disintegrating changes which may take place in metabolism, in contradistinction to the ascending, synthetic, or constructive series of changes which constitute anabolism

**Catabythismus.**—Suicide by drowning. *Catabythismomania* is the form of suicidal mania which seeks to accomplish its end by drowning

**Catalase.**—An enzyme or zymm found in many tissues, and capable of breaking up hydrogen peroxide

**Catalepsy.** See also HYPNOTISM, HYSTERIA, HYSTERIA IN CHILDHOOD, INSANITY, NATURE AND SYMPTOMS (*Cataleptic Stupor*), SPASM (*Hysterical*)

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**DEFINITION.**—Catalepsy is a nervous affection characterised by a plastic rigidity of voluntary muscles, by unconsciousness, including the abeyance of common and special sensibility, and by the abeyance of reflex irritability

**ETIOLOGY.**—Females are disproportionately predisposed to catalepsy, and the period of adolescence is a common age for its occurrence. The affection is predisposed to by any conditions which make for neurasthenia, it has obvious katamemal relations in many cases, and, both in males and females, there are usually some suggestions of hysteria. Very commonly the first attack is occasioned by some mental or nervous incident of a violent nature—a fire, an assault, great religious excitement, a fall, a sunstroke, a sudden affliction, and similar emotional or nervous shocks. Cataleptic affections may appear in association with that form of insanity called stupor, with hysteria, with tetanus, with chorea, and with epilepsy, so as to suggest an etiological relationship

**SYMPTOMS.**—The first attack of catalepsy may be quite unexpected. But the affection is apt to be paroxysmal and even periodic. Even in such cases the patient may be quite well between times, but it is very commonly the case that a more or less sudden giddiness, or headache, neuralgia, hicough, or visual disorders immediately precede a seizure. Therein we observe a first resemblance to epilepsy, and, to appreciate the condition, we must constantly keep that comparison in mind

Whether there has been warning of some minutes or longer, or if there has been none,

the patient is, at the instant, usually aware that she is about to fall. With perhaps some vague efforts to kneel or to reach a seat, she sinks helplessly to the floor or subsides in her chair. Apparently, in a typical seizure, the consciousness of the patient is as it may be in a deep swoon. The last fact that she is aware of is that she has lost control of her muscles, and she appears to know no more until she recovers some minutes or hours or even days later. She has then no recollection of what has transpired meantime. Whether or not there be any isolated mental activity during a seizure, we have no means of knowing.

Meanwhile motor symptoms of a most interesting character have been developed. There is no violent tonic spasm in catalepsy. But there is fixation of the muscles of a nature which is curious and not easy of explanation. An epileptic will crash to the ground as a rule, and then, of course, clonic spasms follow. A hysterical patient will fall with some slight arrangement of her limbs and skirts, and other signs of apparent purpose in her movements or in her pose. But in a typical cataleptic seizure the patient drops off as in syncope, and her muscles become fixed in the first attitude at which she comes to rest. If you try to lift a limb, or rearrange the body generally, you will find that the muscles have stiffened as if in rigor mortis. Then by degrees they gently relax, so as to adapt the pose to the action of gravitation, and the trunk and head and limbs all sink flatter on the ground or couch. At this stage the muscles have reached the characteristic condition of plastic rigidity, the distinctive feature of catalepsy known as *flexibilitas cerea*. The figure is not now cast in metal but in wax, and is plastic under your fingers. It retains any pose which you make it assume. There is no automatic movement of a purposive kind, no lifting of an arm or hand or foot, and there are no spasms. If you raise a limb to a position of strain, the muscles will return that pose for a time, and then by slow degrees will allow the limb to sink to a position determined by gravity.

At this stage the sensibility of the patient will be abolished. The skin is apparently quite insensitive to touch, to heat, to pain, to electricity. Special sensibility is abolished. Reflex irritability is also in abeyance. Even the conjunctival reflex is probably abolished. Respiration is very shallow, cardiac action is slight, and the temperature slowly falls.

A typical seizure has been described. Very commonly there will be important modifications in the symptoms. In particular the consciousness and the sensibility of the patient may not be abolished, though powerlessness and plastic rigidity may have supervened. The reflexes may not be greatly impaired, though common and special sensibility is in abeyance. Hyper-sensibility has been reported as a rare variation

As in other such diseases, there are cases of spurious catalepsy, in some of which symptoms of hysteria are conspicuous, and cataleptoid elements are frequently prominent in allied affections.

**TREATMENT** — During an attack treat the patient as for an epileptic seizure. Put the body in a position of ease, and do not disturb the patient. In the majority of cases spontaneous awakening occurs at the most after a few hours. It is difficult to see what good is served by violent attempts to arouse patients from com. Gentle means may occasionally be tried—a sharp puff of wind on the face, a breath of smelling salts, a pinch of snuff, or even a very mild splashing of cold water. Avoid emetics and painful shocks of electricity. If the attack continues, food may be necessary. If so, pass it into the stomach by the tube, or, in the first instance, empty the rectum and administer a nutrient enema. Draw off the urine if there are reasons for supposing that there is any considerable accumulation. If the patient is comfortably in bed and these measures are adopted, there is no reason for supposing that the cataleptic sleep will be hurtful, even if it is protracted over several days.

After an attack, treat the patient as for neurasthenia. Avoid regarding the case as hysterical. Consider it rather as epileptoid. Try to discover any mental or physical source of irritation, and remedy it if possible. As in chorea, the patient may be greatly benefited by a change of scene, especially if that entails a removal from the chief source of annoyance or excitement. Special regard should be had to pelvic factors in the disease.

**PROGNOSIS** — The prognosis in each seizure is good, and the prospect of immunity from attacks is also good, and increasingly so according to the age of the patient.

**Differential Diagnosis** — In *stupor* there is more or less persistent mental disorder. In *hysteria* the plasticity of the limb receives some added effort of its own. A cataleptic limb is moved from one position to another, and will retain the position in which it is placed for some time. A hysterical limb generally moves. By judicious change of pressure or of direction you may observe the hysterical limb continue to move without your aid, or continue to oppose a strain which you have withdrawn. In *tetanus* there is spasm, and generally obvious signs of pain. In *epilepsy* there are first spasms, and in the later stages the plastic rigidity is absent. In the cataleptic state induced by *hypnotism* the symptoms, in this country, vary considerably, and in general there is, as in hysteria, a feeling on manipulating the limbs as if the patient were following your movements. In death physical signs discoverable by auscultation are wanting, and the *rigor mortis* is not plastic.

**NATURE AND PATHOLOGY** — We have no definite

knowledge of the pathology of catalepsy. The nervous condition previous to a seizure must be held to explain, in a histologist's sense, the nature of the lesion, and it probably is comparable to other related diseases—mania, hysteria, epileptoid states, etc. As to the seizure itself, and its effects, it is important to consider what are the mechanisms which are chiefly involved.

It is obvious that the lesion is partly cortical, and that it involves mechanisms which subserve perception and volition. The coma seems to have features which distinguish it from that of sleep, of induced hypnosis, of epilepsy, or of swoon. The mode of invasion is characteristic, and the muscular accompaniments, and the depth of insensibility. Apparently the lesion is one which specially involves the outward realm of mental mechanisms—those which subserve muscular activities. The loss of voluntary movement is the prelude to the curious phenomenon which persists—the plastic rigidity in voluntary muscles. The invasion of consciousness would seem to be somewhat similar to what occurs in certain epileptiform attacks (not epileptic) in which the spasms of voluntary muscles initiate an attack in which consciousness is thereafter lost. In catalepsy there is paralysis of voluntary movement, followed by paralysis of consciousness. The most interesting phenomenon, however, still remains—the balanced and co-ordinate muscular contractions which determine rigidity of the limbs, and their persistent sensibility to pressures which determines their plasticity. That phenomenon undoubtedly suggests, as Gowers has pointed out, an affection of the muscle-sense organs. Affluent fibres from the muscles, fascias, and joints, and corresponding motor tracts, complete a reflex arc whose apex is in the spinal cord. These mechanisms subserve the function of unconscious balance and pose. In normal life, however, that reflex arc has an extension upwards to the cortex, which makes possible a voluntary control of its activities. In catalepsy the higher activity is in abeyance, and the lower mechanisms are active in excess. The rigidity then is simply a secondary, positive sign of the removal of conscious control, and the plasticity of it is part of its normal function—to adapt pose to stress. But the extraordinary thing is that this function *should* persist when others have been abolished—an isolated and excessive activity in a general condition of profound coma. We are therefore led to conjecture that there is something very like an epileptiform seizure in catalepsy, and that the motor convulsion, if there is one, is a convulsion in this mechanism to which we have referred. The absence of obvious spasms, either tonic or clonic, signifies only that the convulsion is not in a mechanism of large movements, which we already know. The mechanism referred to is a fine-adjustment mechanism. Its movements are all small. It would be instructive to

know if there are any cases in which, with prolonged unconsciousness, there are recurrences of the characteristic muscular phenomena. Some of the protracted cases may signify the passing from one fit to another. In any case, as all authorities are agreed, the affection is in some sense epileptoid.

**Catalysis.**—The effect produced upon a body or substance by the presence or contact of another substance, the latter being termed the catalyser and showing in itself no change, it is in this way that enzymes are believed to act (*e.g.* in fermentation), it has been supposed that the catalyser does not really initiate a reaction, but merely hastens it.

**Catamenia.** See MENSTRUATION AND ITS DISORDERS (*Terminology*)

**Catapasm.**—A dusting-powder

**Cataphasia.**—A disordered state of speech, a word or phrase is repeated or affirmed (*κατάφασις*, affirmation) over and over again, either as an answer to a series of questions or apart from conversation.

**Cataphoresis.**—That action in which galvanism aids a substance or solution to penetrate the tissues acted upon, a sort of electric osmosis. See TEETH (*Electro-Cataphoresis*).

**Cataplasm.**—A poultice, occasionally, a plaster. See PRESCRIBING

**Cataplexy.**—Mesmeric sleep in animals, shamming death, or a temporary paralysis.

**Cataptosis.**—The sudden feeling of a patient affected with epilepsy or apoplexy.

**Cataract.** See also DIABETES MELLITUS (*Complications, Affections of the Eye*), EYEBALL, INJURIES OF, GLAUCOMA (*Causes, Cataract-Operations*), OCULAR MUSCLES, AFFECTIONS OF (*Nystagmus*)

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TECHNICALLY, the name cataract is applied to any intransparency of the crystalline lens. Popularly, only those lenticular intransparencies which by their distribution and their density cause more or less considerable impairment of visual acuity are looked upon as cataracts. It is also

common to talk of cataract developing when the intrasparencies in the lens are of the nature of progressive degenerative changes

From a diagnostic point of view it is often important to distinguish between the "stationary" varieties of cataract in the technical sense and the "progressive" forms. The former are also called "partial" cataracts, whilst the latter eventually become "total," or practically total, cataracts

**FORMATION OF CATARACT**—The loss of transparency which causes a progressive cataract is invariably the result of some degenerative change. The extent, moreover, to which these degenerative changes go leads to different appearances. There thus come to be distinguished a number of different varieties of total cataract. Only comparatively few of these varieties have any practical importance. The importance which may attach to them practically has reference to the selection of the most suitable operation for their removal, and consequently also to some extent to the prognosis of the result of operative interference

A number of terms are in use to designate the stage at which a progressive cataract has arrived. Thus in any particular case the cataract may be "incipient," "advanced," "ripe," or "over-ripe." The meaning of the first two terms is self-evident. A cataract is ripe when the whole of the lens has lost its normal transparency. As a rule the selected central portion of the lens, which is met with after the age of about thirty, and which forms as ago advances a greater and greater proportion of the whole lens, the so-called "nucleus" of the lens, does not at first participate in the loss of transparency. It is therefore the totality of the surrounding and more superficial or "cortical" portion of the lens which is the site of a degeneration causing the transparency to be lost

When the whole "cortex" has become intrasparant, then the cataract is said to be ripe. The term "ripe" has reference to the circumstance that the lens proper is then readily removed out of its capsule like a ripe nut from its shell.

The further progression of the degenerative changes which have caused the total intrasparancy lead to over-ripe cataract. Thus the cortex may become liquefied or cretaceous. In addition, the lens capsule may in part (over an area corresponding to that left uncovered by a semi-dilated pupil) participate in the loss of transparency or the suspensory ligament of the lens undergo degenerative changes. These latter changes are of importance from the point of view of treatment

When once the lens cortex has lost its transparency, it is extremely rare that the transparency is regained. Practically speaking, there is therefore no cure for cataract in the

sense that the eye can recover its normal condition. Absorption of the altered lens substance may, however, take place spontaneously. In many cases this can be induced by surgical interference. When the intrasparant lens is thus got rid of by absorption, the obstacle to the formation of retinal images is removed and sight is restored. The optical condition of the eye is, however, different from what it was with the transparent lens *in situ*. Generally the new condition is one which admits of less sharp retinal images. Sharp vision can then generally only be got by means of a glass. The object of most cataract operations is to get rid of the intrasparant lens. In all cases of total cataract this is necessary.

Cataract may be either traumatic or idiopathic. However arising, it is essentially due to some defect in the nutrition of the lens fibres

**TRAUMATIC CATARACT** is either the *direct* result of some interruption in the integrity of the lens capsule, or the *indirect* result of injury to other parts of the eye. In the first case the loss of transparency of the crystalline lens is due to its coming into direct contact with the aqueous humour. In the second case the injury to deeper parts of the eye leads to intraocular changes, with which are associated changes in the character or in the amount of the nutrient liquids which percolate the lens

In order that the lens may remain transparent, it is necessary that the lens capsule should be normal. The smallest breach in continuity of this membrane leads to intrasparancy.

A cataract caused by a wound of the capsule makes its first appearance very soon, in a few hours at most, after the injury, and afterwards develops more or less rapidly according to circumstances. Very rarely, and only in cases in which the laceration of the capsule is very minute and has rapidly cicatrized, the intrasparancy of the lens which follows may remain partial, or even slowly disappear altogether. This rare result is only met with too in young children

The laceration of the lens capsule is usually caused by a perforating wound of the eye. Occasionally a sharp blow on the eye may cause a rupture of the capsule, and the result is similar, viz an immediate loss of transparency of the lens.

Such cases of capsular injury, either by perforation or rupture, cause, then, what may be called a direct traumatic cataract. In other injuries to the eye unassociated with injury to the capsule, the cataract which may follow is "indirect." An indirect traumatic cataract only makes its appearance weeks or months after the injury

**IDIOPATHIC CATARACT** again may be either "primary" and uncomplicated, or "secondary"

to some other disease which of itself has caused, or is likely to cause, serious interference with vision

Of the primary cases, uncomplicated so far as any other defect of the eye is concerned, two groups may be recognised. The first group, fortunately by far the larger, includes all the cases in which there is no evidence of any general condition of malnutrition. In the second group there may be more or less reason, on the other hand, to connect the loss of transparency in the lens with some such condition as albuminuria, diabetes, etc.

Idiopathic cataract, when primary and occurring in an otherwise healthy individual, is most common after the age of sixty. On this account it is usually known as *senile cataract*. There is, however, no necessary connection between this variety of cataract and other evidences of senility. In many cases there seems to be a more or less evident hereditary tendency to cataract. On the whole, it would appear, too, as if the younger the individual affected with cataract, the more evident was the hereditary predisposition.

Apart from heredity, which is by no means always traceable, there does not appear to be any general state associated at all intimately with the appearance of cataract. All that can be said is that, on the whole, there is a tendency increasing with age for the nutrition of the lens to become impaired, and that this tendency is greater in some families than in others. Probably the structure of the lens itself predisposes to defective nutrition. The lens is an epithelial structure, which, as time goes on, becomes more and more compressed and sclerosed in its central portion. This compression results from the continual growth of new lens fibres which go to form the outer layers, whilst the old, effete tissue, not being removed, gradually accumulates. The oldest and most horny portions of the lens, instead of being rubbed off, as in the skin, are thus collected in its centre. This arrangement of itself seems to predispose to difficulties of nutrition. These difficulties are more insuperable in some individuals than in others, altogether independent of anything but mere local conditions. Whatever be the true and complete explanation, it is of practical importance to recognise that the term *senile* as applied to cataract has only a restricted sense. The appearance of cataract is therefore not necessarily, or even commonly, an indication of any general breakdown.

Most cases of *senile cataract* are cortical cataracts, i.e. the loss of transparency which takes place is limited to the cortex. In very old people the lens nucleus may become clouded, and then usually assumes a dark brown or almost black colour. This is sometimes, and especially when the colour is very dark, called black cataract (*cataracta nigra*). A better

term is *nuclear senile cataract*. Although in the aged the nucleus of the lens comprises almost the whole of that structure, so that very little cortex remains, the true nuclear senile cataract not being such an essentially degenerative change, a nuclear cataract does not cause such a complete loss of vision as is caused by an ordinary ripe senile cataract.

**DIAGNOSIS.**—Mere inspection of the eye reveals a greyish opacity in the pupil. The pupil itself is freely movable, contracting as light falls into the eye, and dilating when the eye is shaded from the light. The history, too, is one of gradually increasing blindness until the details of objects have become indiscernible, and only the movement of large bright objects in front of the eye, or it may be only the difference between light and darkness, can be made out.

The earlier stages of cataract, as well as the differential diagnosis between visual defects due to intran transparencies of the lens or to other causes, can only be made with the ophthalmoscope. Any intran transparency in the dioptric media (the vitreous, lens, aqueous, and cornea) appears in the disc of *fundus reflex* (see "Eye, Examination of") as a dark area interrupting its continuity. When dark spots are seen in this way, the diagnosis of the site of the intran transparency has next to be made. A preliminary examination of the cornea and aqueous made by *oblique illumination* enables us to see how far any intran transparencies in these parts contribute towards the appearances seen with the ophthalmoscope. If there are no corneal nebulæ and the aqueous is clear, these parts of the eye can be excluded, and the question then is whether the dark areas interrupting the fundus reflex are due to intran transparencies in the lens or vitreous humour. Opacities in the vitreous are often movable, i.e. move independently of the eye when once set in motion by eye movements. Lens opacities are, on the other hand, stationary. The differential diagnosis is usually easily made, although in some cases of opacities lying far forwards in the vitreous there may be some difficulty.

*The pupil in old people* often appears grey or greenish grey. It presents then a very different appearance from the dark, black pupil seen in earlier life. This grey appearance is due to light reflected from the anterior surface of the lens. In young people the superficial layers of the lens have a refractive index very nearly equal to that of the aqueous. Consequently little or no light is reflected from the surface of separation between these two media. The greater hardness of the older lens, with its consequent greater refractive index, causes considerable reflection from the lens surface. This reflection is sometimes so great as to give the appearance on mere inspection of cataract. Inasmuch, too, as old people often complain of failing sight from other causes than cataract, it is important not to mistake the condition described for

cataract. All that is necessary to make the differential diagnosis is the *dioptric* test with the ophthalmoscopic mirror. When no cataract exists, the fundus reflex, so far as the lens goes, is seen undisturbed throughout.

*Sensile cataract* is mostly met with in both eyes. Often the condition first begins in the one eye alone. The first appearances are only seen with the ophthalmoscope and with the pupil dilated. They are generally those of radiating peripheral intran transparencies, the so-called *striae*. The stage of peripheral *striae* may last for many years without appreciably advancing. Generally, however, the *striae* become continually more numerous, larger, and confluent. Only small chunks of clear cortex then remain. Often this gives rise to a multiplication of the images of objects seen—what is called *polyopia*. Sometimes, however, the vision remains amazingly good, though the changes are often accompanied by alterations in the optical condition of the eye. Weaker reading glasses are then often used, and the distant vision is improved, it may be by concave glasses, or if previously convex ones have been required, they may no longer be necessary.

The further confluence of the separate areas of intran transparency leads to the whole of the cortex being involved, and then generally pretty soon to the cataract becoming ripe. The period occupied in ripening, however, varies very much. As a rule, from the time the *striae* first become evident till the whole cortex is intran transparent, there is an interval of time of from two to three years. But there are frequent exceptions to this, and mostly in the direction of a prolongation of the period of development. It is therefore not safe in any given case to venture upon too definite a prognosis as to the date when the cataract will be ripe.

There is often a very serious degree of blindness long before the cataract is ripe. The bearing of this fact upon the question of treatment is afterwards considered.

The stage at which operation for cataract is most satisfactory is the stage of ripeness. Interference is on the whole less satisfactory before this stage has been arrived at, and still less satisfactory when there is any marked degree of over-ripeness.

The most important point which has to be inquired into in the case of a ripe cataract is as to whether or not the cataract is uncomplicated. A ripe cataract, whilst it leads to the loss of any sense of form, so that surrounding objects are not seen, does not materially interfere with the sense of light. Consequently an individual with a ripe cortical cataract has a good appreciation of differences in degree of illumination. The light from an ordinary candle flame in a dark room can be seen, and the difference at once recognised when it is shaded by the observer holding his hand in front of it, so as to prevent its rays falling on the patient's eye, or the

difference caused in illumination by reflecting into the patient's eye the light of a gas jet turned down to almost its lowest point and then removing the reflection is at once appreciated. One circumstance, then, which would cause one to infer that the eye was otherwise healthy, notwithstanding the existence of a ripe cataract, would be the possession of a keen perception of light. On the other hand, a defective light sense must cause suspicion of deeper disease. When the light sense is very defective there can be no doubt of the existence of complication. And this is, of course, still more the case when the patient is unable at all to distinguish light from darkness. This condition is a complete contra-indication to operation, as no sight could then be restored by removing the cataract.

Another point which has to be tested is in the cataractous eye called the *projection*. Although unable to distinguish ordinary objects, an individual with ripe cataract, if the eye be otherwise healthy, should nevertheless have a fair idea of the direction from which a bright light comes. Thus the direction of a candle flame in a dark room, held close to him above or below or to either side of the face, can be made out. Although the intran transparent lens is not capable of forming sharp images, it is able, even in cases of ripe cataract, to so far collect the rays, whilst scattering most, that a maximum illumination of the retina takes place in and around the place at which, under normal conditions, the image of the flame would be formed.

**CONGENITAL CATARACT**—In addition to the forms of cataract already enumerated, there is a whole series of juvenile and congenital cataracts. These may be complete or partial, cortical or nuclear.

It is most important to recognise and to treat congenital cataracts as early as possible if they are either complete or so extensive and dense as to cause a serious impediment to vision. If they are not removed within the first few months after birth, the resulting vision is rarely good. Cataract developing in eyes which have once acquired good sight may remain for an indefinite period without interfering with the possibility of sight being restored on its removal. The immediate result of a successful operation in such cases is in fact the complete restoration of the sight, even if the cataract may have been there for forty years or more. There is no amblyopia from disuse. It is otherwise, however, where the early development through practice, which is necessary for the perfecting of the eye as a seeing instrument, has been rendered impossible. Later operation will admit of a certain degree of sight being acquired. But the acquiring of sight under these conditions is a slow, laborious process, requiring often much training. It always stops short of perfection—often much short of it. Operations in the early months of life give the best chance. The necessity for



early operation is the main practical point to keep in view in connection with such cataracts. Their diagnosis presents no difficulties, and the differences met with in different cases are only of scientific interest. They are almost invariably bilateral.

A complete cataract existing in one eye only of a child is almost certainly of traumatic origin. A careful examination of the cornea will usually in these cases reveal a scar, often a very faint one, owing to the rapid tissue changes of childhood. The scar indicates where the perforation of the eye took place. Curiously enough, parents and others who bring young children with traumatic cataract long after the accident which caused it often deny that any accident has taken place. They usually admit it, however, when it is pointed out to them that the scar is visible. Sometimes, as, for instance, when the penetration has been made with the point of a fine needle or a fine pair of scissors, the parents may actually be ignorant at the time that so serious an accident has occurred.

One of the most frequent forms of juvenile cataract is what is called *lamellar cataract*. This is a partial, stationary cataract. It is either congenital or developed very early in life. In lamellar cataract only some of the lens fibres, which are arranged in lamellae, are intransparent, hence the name. The defect of vision which this variety produces varies in degree according to the position and number of the intransparent lamellae. The pupil shows only a moderate degree of opacity, sometimes, indeed, so slight that it is difficult to diagnose the condition without the ophthalmoscope. The ophthalmoscopic appearance is characteristic. If the pupil be dilated it is found that a disc-shaped darkening of the fundus reflex occupies a central area of it. The shaded area varies in extent, appears darker at its margins, and is surrounded by a clear zone. Often the circular line corresponding to the margin of the opacity is broken by little protrusions, which on closer inspection are found to be caused by more peripheral intransparencies, which, as they embrace the edge of the main opacity, have received the name of "riders."

Lamellar cataract is rarely noticed before schooling has begun. Then it is found that the child's sight is less acute than normal. Objects in order to be seen are held closer to the eyes than under normal conditions. This attracts attention, and is usually supposed to indicate the existence of myopia. Often, indeed, and no doubt as the result of this too close application of the eyes, the condition actually does become complicated with myopia. Anything, in fact, which causes the persistent use of the eyes in early life for the seeing of objects which lie too near to them is apt to give rise to myopia.

The question which has to be determined in all cases of lamellar cataract is whether or not

it is desirable to operate. As the operation consists of removing the lens in some manner or other, any power of accommodation must necessarily be lost after operation. The question to be considered then is whether the improvement in vision which the operation will cause is sufficient to counterbalance the risk of operation, the necessity for wearing cataract glasses, and the loss of accommodation. Obviously, there must be some limit for which the advantages and disadvantages are pretty equally balanced. This limit will vary, too, according to the individual views of different operators. Some even go the length of operating on one eye only, leaving the other untouched, so as to retain as far as possible both advantages, that of increased visual acuity in the operated eye, and retention of accommodation in the one which remains unoperated upon. This, of course, is done at the expense of binocular vision. It is rare that such a practice is to be recommended. Indeed, it should only be resorted to when one eye is considerably worse than the other, and at the same time the occupation of the individual makes it seem advantageous. In this respect each case must be judged on its merits.

A good practical rule to be guided by is to recommend operation when the visual acuity is less than  $\frac{1}{10}$ , and to advise the patient to have nothing done where the acuity is greater than this limit. Here, again, however, one must to some extent be guided by the wishes of the patient, when matters have been properly explained to him or his parents, as well as by the nature of the occupation which he follows or wishes to follow.

Lamellar cataract is almost invariably bilateral. It seems to stand in some relation to rickets, though in most cases the manifestations of rickets are not particularly pronounced. Often there is an absence of enamel on the teeth, especially the canines. Children with congenital cataract in any form are occasionally of more or less markedly weak intellectual development.

*Treatment of Cataract*—Nothing is known to have any definite effect in checking the progress of cataract. The degenerative process seems as a rule to be so intimately associated with difficulties of nutrition depending upon the natural structure of the crystalline lens, and so rarely dependent upon general conditions of health, that it is unlikely that the progression of cataract could be influenced by general treatment. It is conceivable, no doubt, that the conditions of nutrition might be modified to advantage by local treatment. Hitherto, however, any treatment which has been adopted to dissipate cataract has been by secret remedies. The favourable results claimed for such remedies have never been substantiated. It is even doubtful if any ordinary use of the eyes in any way influences the process. This being the

case, it is hardly fair to impose upon one's patients, as soon as the beginning of cataract is diagnosed, all sorts of restrictions instead of allowing them to have the full use of their eyes as long as possible. Excessive, continuous reading, especially with a bad illumination, may perhaps be undesirable.

When cataract has advanced sufficiently to cause some marked interference with sight it is sometimes possible to get some temporary improvement by the use of a weak mydiatic or miotic or by suitable glasses. In many cases of advancing cataract it is found that patients complain of seeing very badly when facing the light, or generally, when the illumination is strong. In reading, for instance, they prefer sometimes an illumination which is otherwise unsuitable owing to its being too feeble. The reason for this is that when the pupil is small the total area of the portions of the lens which remain clear is insufficient to give a bright enough retinal image. With a semi-dilated pupil, however, the number of rays going to form the image is greater. And if the more eccentric pupillary portions of the lens are relatively free from intraparties, the proportion of effective rays may increase at a greater ratio than the diminution in illumination, which causes the pupil to become less contracted. A still greater advantage may under these conditions be got by causing an artificial dilatation of the pupil. The greater number of rays thus admitted to form the retinal image is not counteracted by the diminished illumination necessary to admit of a natural dilatation of the pupil. This artificial dilatation of the pupil is best secured by the use of a weak solution of sulphate of atropine ( $\frac{1}{4}$  gr to  $\frac{3}{4}$ ), which may be applied daily, or even once every second day. In advancing cataract this should always be tried. If it proves of use it may then be safely indefinitely continued. It is comparatively seldom, on the other hand, that a miotic by causing an artificial contraction of the pupil is of use in cataract.

Patients with cataract often suffer too from a disagreeable degree of dazing. This is caused by the scattering of many of the rays which enter the pupil. These scattered rays, mixing with the regularly refracted ones, disturb the clearness of the retinal images, and produce this disagreeable sensation. It is for this reason that cataract patients rarely face the light, or, when they do, walk with their heads bent forwards. They frequently, too, shield the eyes by holding their hands at the side of the face, or by shading the eyes in some other way. Sometimes spectacles in the form of stenopæic slits cause improvement in vision by diminishing the amount of this scattering.

When the cataract in one eye is further advanced than in the other, the same scattering of rays in the worse eye causes the vision with

both eyes to be less perfect, and associated with more discomfort than when the better eye is used alone. It is then advisable to allow them to read with an obscure screen in front of the worse eye.

Another expedient which may be resorted to when the sight is insufficient to permit of reading with the proper correction at an ordinary distance is to give up any attempt at binocular reading and use the better eye alone. It is then provided with a glass of sufficient strength to focus it for a distance of 6 or 4 or even 2 inches instead of for 10 to 14 inches. The retinal images thereby obtained are correspondingly larger, and thus reading may be continued for a longer time than is otherwise possible. Reading with a hand magnifying-glass also serves the same purpose.

The various operations performed for cataract may be classed under three heads—(1) Reclination, (2) Discussion, and (3) Extraction.

*Reclination* or *couching* does not seem to be practised in civilised countries. It is a very old operation, and the one which was at one time universally employed. The operation consisted in pushing a needle, entered through the sclera, forwards between the lens and ciliary body, and then, by raising the handle, pushing the cataractous lens backwards into the vitreous. This operation, though producing necessarily, in uncomplicated cases, an immediate restoration of sight, was abandoned owing to the frequency with which it was followed by inflammation or glaucoma. It must be remembered, however, that reclination was performed exclusively in the pre-antiseptic period. Indeed, not only were no antiseptic precautions taken, but some of the descriptions given of the operation make it clear that it was customary for some operators to dip the cataract needle into, or smear it with, substances which were more likely than not to be septic. The risk of reclination nowadays could certainly not be nearly so great as it formerly was. Nevertheless, as a surgical proceeding, it is inferior to discussion or extraction.

*Discussion* consists in opening the anterior capsule with a fine, sharp cataract needle, and then passing the needle deeper into the substance of the lens and stirring it up. The aqueous humour is then brought into direct contact with the substance of the lens, which is macerated, driven forward in flocculent masses into the anterior chamber, and absorbed. The absorption takes place through the channels, at the angle of the anterior chamber, by which the aqueous leaves the eye. If the discussion has been free, flocculent masses of macerated lens matter readily escape out of the capsule and lie in the anterior chamber. There they gradually become lessened in size as absorption slowly proceeds.

In performing discussion care must be taken

to make a sufficiently large opening in the capsule. When the lens is stirred up the swelling which takes place as it becomes macerated leads to an extrusion of some of its substance through the capsule wound into the anterior chamber. If the wound in the capsule is not large enough, as is, for instance, the case if the capsule has been merely punctured before the lens has been stirred up, the intracapsular swelling which follows may cause more or less irritation of the eye due to increased intraocular tension. Apart from this, however, absorption does not under these circumstances take place so readily, or it may stop altogether. The operation has then to be repeated.

The cataract needle in performing a discission should be passed very obliquely through the corneo-scleral margin into the anterior chamber, thus forming a valvular opening. After having reached a little beyond the middle of the chamber, the capsule should be lightly cut by raising the handle of the instrument till the point of the needle comes in contact with it. The needle point is then drawn over the surface of the capsule, and made to tear a linear aperture in it. The instrument is then slightly withdrawn till its point lies again free in the anterior chamber, then again pushed in sufficiently to enable another section of the capsule to be made in the same way, but in a direction crossing the first at as great an angle as possible. In other words, the capsule should be opened by a pretty free crucial incision which does not pass too deeply. After this is done the needle is pushed into the lens and stirred about. Care must be taken not to perforate the posterior capsule, which may readily be done towards the periphery where the lens is thinner.

The operation of discission done with an aseptic needle causes very little reaction. Absorption, however, takes place very slowly, several months elapsing before the pupil is clear. Occasionally after three or four days glaucomatous symptoms may develop. In children this is often accompanied by sickness, and the eye becomes congested and painful. It is then necessary to relieve the tension by extracting some of the macerated lens from the eye. This is done through an opening made at the corneo-scleral margin with a bent keratome. The lens matter is removed through this incision along a curette (a small grooved instrument made for this purpose), with which the outer lip of the wound is depressed, and which is introduced a short way into the anterior chamber.

This operation of linear piecemeal extraction is very often made to follow a discission even when no symptoms of irritation have supervened. The removal of the cataract is thus hastened. When properly performed, and with due antiseptic precautions, it is a perfectly safe proceeding.

Operation by discission with or without a subsequent linear extraction is only suitable for cataract in young people. Different operators, however, recognise different limits of age, after which discission should not be done. It is, generally speaking, not advisable to operate in this way when the patient is more than twenty-five. Too much irritation is otherwise apt to be caused by the harder central portions of the lens. In any case the later a discission is done the more necessary is it to follow it up with a linear extraction. As a rule it is better to perform the extraction at once, and not subject the patient to two operations.

Discission followed by linear extraction is the most satisfactory operation for lamellar cataract in children. It is also the operation generally performed for traumatic cataract in young people.

*Extraction of the opaque lens as a whole from the eye is the method of operating which still remains to be described.* There are several different methods of extraction in use. These methods mainly differ in minor details. The general principle of cataract extraction is to remove from the eye as completely as possible the whole of the intransparent lens in a manner which is conducive to rapid primary union of the external wound made for the purpose. The wound must not only occupy a position which does not interfere on coadaptation with vision, but it must be so placed that the edges lie in good apposition after the cataract is removed, and are also readily nourished. The site usually chosen for the wound is therefore the corneo-scleral margin. Some operators, however, carry the incision more or less decidedly into the cornea.

The operation of extraction is performed with a long narrow knife, the so-called *Graefe knife*. The incision into the eye is preferably made along the upper corneo-scleral margin, where it is afterwards mostly covered by the upper lid. In making the incision the knife is entered at the outer side at a point in the corneo-scleral margin on about a level with the horizontal tangent to a semi-dilated pupil. This is what is generally called the *puncture*. The knife is then carried rapidly across the anterior chamber, and a *counter puncture* made in the inner and upper corneo-scleral margin at a point just opposite to the puncture. The incision should lie throughout in the corneo-scleral margin, and should be made with as little sawing motion as possible. The wound should, in fact, be as clean-cut as possible, and occupy about one-third of the corneo-scleral margin. If the extraction be combined with iridectomy a portion of iris is next excised, the iris being drawn out through the incision either with iris forceps or hook, and then snipped across with a pair of sharp iris scissors. When the hook is used this can be done without causing any pain. After replacing the edges of the wound (coloboma) in the

iris, should they be caught at all in the external opening, the capsule is ruptured with the cystotome. The incision of the capsule is made in different ways by different operators. The main point, however, to attend to is that it should be sufficiently free. This is best effected by scratching the capsule several times, so as to make sure that the cystotome is not merely moved on the surface of the lens below the capsule without enlarging the opening first made in that membrane. The cataractous lens is then caused to become engaged with its upper edge in the external wound. This is done by pressure on the lower half of the cornea. When it has thus become engaged, further pressure, followed by gliding the scoop used for the purpose upwards, causes the lens to escape out of the eye. Particular care must next be taken to replace any iris that may be caught in the ends of the incision. This is best done with a soft caoutchouc or tortoise-shell *repositor*. In the process of delivery it usually happens that some of the soft cortical portion of the lens is rubbed off and remains in the eye. Subsequent gentle pressure on the cornea, which is most conveniently made through the lid, enables one to get rid of this. The pressure causes the external wound to gape slightly, and at the same time rubs the cortex through it. Before applying any dressing it is necessary to make sure that the lips of the wound are in good apposition, and that they do not entangle any capsule or cortex. The repositor should be run along between them, so as at the same time to flatten out any flap of conjunctiva which may have been cut in making the incision.

When extraction is performed without iridectomy, what is called the *simple* as opposed to the *combined* extraction, the external wound has to be made slightly larger. The pressure, too, must be rather greater, as the lens has to pass in front of the iris, which necessitates also making the wound gape to a greater extent. In this operation cortical remains are removed with the curette, and not by pressure, after the iris has been carefully replaced, should the pupil not of itself have recovered its circular shape.

**THE QUESTION OF IRIDECTOMY OR NO IRIDECTOMY**—Whether the simple or the combined operation is to be preferred is one upon which much has been said, and one upon which different surgeons hold different views. The advantages of the simple operation are, that no wound is made in the iris, that the cosmetic effect of a round pupil aimed at in this operation is better than that of a coloboma even when the coloboma is made upwards, and that peripheral attachments of the iris to the inner surface of the external wound are less common. The disadvantages are that cortical remains are not so easily removed after the delivery of the lens, and that prolapse of the iris into the wound is

more apt to take place than when iridectomy is done. In addition, the circular mobile pupil aimed at is by no means always got. Further, the simple operation is not so easy, and on this account alone not to be recommended to inexperienced operators. There is no difference in the result, so far as the acuity of the restored vision goes, which is got by the simple as compared with the combined extraction. The simple extraction is in other respects, however, the more ideally perfect one.

In making the somewhat larger section which is required in the simple extraction there is some difficulty often in preventing the iris from falling in front of the knife as the anterior chamber empties. The blade must therefore be kept pushed well forward, raising as it were the cornea from the chamber. A notice should also be used immediately before making the section, so that as soon as the iris is returned the pupil may be contracted. The eye should be examined from half an hour to an hour after the operation is completed. If the pupil at this stage is found to be contracted and quite circular, there is practically no danger of iris prolapse taking place. If, on the other hand, it is not circular, but hitched up towards the wound, the round movable pupil aimed at will not be got, and there is in addition a strong probability that healing will be impeded and more or less unnecessary risk run by prolapse. Under these circumstances the best thing to do undoubtedly is to perform an iridectomy at once, and therefore convert the simple into the combined extraction. This is very much more satisfactory than any treatment of prolapse at a later stage in the healing process. In the experience of the writer this abandonment of the intended simple extraction is required in 10 per cent of cases in which it is begun. In this respect, however, individual differences must of course exist.

This is hardly the place to discuss fully the relative merits of the simple and combined operations. Some surgeons perform exclusively the former, and some exclusively the latter, whilst others again make a selection of cases which appear to them most suited for the one or other method of operation.

The writer, after a very extensive experience of both operations, would, however, offer the following advice to less experienced operators—

- 1 Do not attempt the simple operation at all until sufficient experience of the combined operation has been obtained by the performance of two or three hundred extractions.

- 2 In the first simple extractions undertaken, select cases of fully ripe cataract in, comparatively speaking, young individuals, say up to the age of sixty or sixty-five.

- 3 Do not hesitate to abandon the simple extraction aimed at whenever the conditions above described indicate a risk of more or less troublesome prolapse.

It occasionally happens that when the attempt is being made to remove the lens by pressure on the cornea after the capsule has been ruptured, vitreous humour presents at, or escapes by, the external wound. Pressure has then to be abandoned and the lens has to be taken out with the *vectis*. This instrument, which consists of a fine steel loop, is inserted behind the lens by being gently forced past its upper margin with a slight side to side movement. As soon as it has slipped far enough back it is pushed downwards so that the lens comes to lie upon it. The *vectis* is then slowly withdrawn with the lens, which is at first kept pressed up against the back of the cornea until it has so far escaped from the eye that it readily comes away altogether. After this proceeding, if the lips of the wound do not lie in good apposition, the iris scissors should be used to cut across any vitreous which protrudes.

This accident may occur from want of care in the use of the cystotome or by too great straining on the patient's part. The suspensory ligament is then ruptured. In some cases, especially where the cataract is over-ripe, it may be impossible to prevent. As it is a complication which cannot always be foreseen, a *vectis* should always be at hand when performing a cataract extraction.

A considerable loss of vitreous may take place without impairing the result of a cataract operation. Yet there can be no doubt that with the loss of vitreous, which is never replaced, the future of the eye is not so secure.

Another complication which often calls for some modification of the method of operating is the co-existence of capsular cataract. When the capsular thickening is not very extensive the extraction may be performed in the ordinary way. With a large, dense, capsular cataract, however, it is best to extract a portion of the capsule instead of merely incising it before the lenticular cataract is removed. This is done with capsule forceps, of which there are various patterns. Extraction of the portion of capsule must be done carefully by gently moving the forceps from side to side as it is being withdrawn after having obtained a good hold of the membrane.

#### AFTER-TREATMENT OF CATARACT EXTRACTION.

—Though not absolutely necessary, it is a good precaution to take to keep the patient in bed for two or three days after operation. This is the best way of as far as possible securing rest to the eye. When this treatment proves very irksome, as it is apt to do in very stout or in diabetic individuals, it is better not to insist upon it.

With regard to dressings different ideas obtain. Some surgeons do not tie up the eye at all. Others apply some sort of dressing and bandaging, which is retained for a varying time afterwards. Others again tie up both eyes

Cataract extraction is an operation in which, given a healthy eye, rigid antiseptic precautions as regards the instruments used, and a certain amount of skill, and particularly quickness in performing the operation, the tendency to healing is fortunately quite remarkable. On this account it is practically impossible to justify any claim of superiority for one method of after-treatment over any other which is not altogether irrational. The rule in such a case should be to err rather on the safe side. It is therefore probably on the whole best to tie up the eye which has been operated on for a few days. Three or four days is sufficient. The dressing, too, may be changed once a day and the eye examined at each dressing. The surrounding light should also not be excessive where it is possible to control this. The tying up of the other eye does not seem to secure any greater rest to the one operated upon, as the patient instinctively keeps it closed for the first few hours until healing is sufficiently advanced to prevent movement giving rise to any pain. As in many cases, too, such treatment is found to be very trying to the patient, it is better to avoid it.

The only exception that should be made as regards tying up the eye after extraction is when there is an evident risk of infection from local sources, as there is, for instance, particularly in patients suffering at the same time from blepharitis of the lacrimal sac. In these cases it is better to leave the eye altogether uncovered. Where this complication exists it is also a good plan to dust freely finely powdered iodoform on the wound immediately after the operation is completed. The purulent secretion should subsequently be carefully removed from the lids two or three times in the twenty-four hours by irrigation with an antiseptic lotion. In view of the risk of infection, too, the operation for a cataract which is complicated in this way should be done as quickly as possible. It is especially important that no prolonged effort should be made to remove cortex, as every moment and every movement of the eye may be supposed to increase the chances of micro-organisms settling in the wound.

As a general rule the anterior chamber is found to be re-formed in a few hours after extraction, not infrequently indeed even within one hour. This means that after so short a time sufficient agglutination has taken place between the lips of the wound to admit of the retention of the aqueous humour. The cleaner the section and the more perfect the apposition the more rapid is this primary union. Hence the importance of a sharp knife and the absence of sawing movements in making the section, as also the final cleaning of the wound.

Very little reaction takes place as a rule. There is usually a little congestion merely of the vessels in the neighbourhood of the wound.

This congestion increases slightly during the three or four days that the wound takes to consolidate. Any further reaction is due to various causes. The most important is infection of any kind during operation or from local sources before healing has advanced. Other causes are the irritation of cortical remains and antiseptics introduced by instruments. A superficial conjunctival irritation often follows from too prolonged tying up of the eye or from the too frequent use of antiseptic lotions.

*Of the Results of Infection* the worst and most dreaded is primary suppurative of the cornea. This generally leads to complete destruction of the eye. Occasionally it stops short of this either as the result of the local vital reaction or of energetic and timely antiseptic measures. An infection of this kind usually shows itself within the first forty-eight hours after operation, often indeed within the first twenty-four hours. The patient complains of pain, the lids are red and swollen, and on inspection of the eye a greyish infiltration is seen stretching towards the centre of the cornea from the wound. An attempt should be made to check this at once as soon as it shows itself. This may be done by applying the thermo-cautery along the wound or by the use of freshly prepared chlorine water or of pure liquid carbolic acid painted on the wound with a fine camel's-hair brush. The wound may then be dusted with finely powdered iodoform and all dressings removed from the eye. If the patient be old and feeble it is well also to use stimulants freely.

Most cases of primary suppurative occur from infection which has its origin in the conjunctiva or tear sac. The cases most likely to go wrong in this way are those complicated with blenorrhoea of the tear sac, especially when this is associated with blepharitis. When there are no such evidences of local infection it is nearly always the case that micro-organisms have been introduced by the instruments. In pre-antiseptic times extraction failed from this cause in a very much larger proportion of cases than now. Yet even at the present time probably no surgeon is able to steer free of corneal infection altogether. No doubt, therefore, there are exceptional cases in which the vitality of the cornea is insufficient to withstand infections which under ordinary circumstances would be counteracted. With rigid antiseptic precautions, however, not more than one eye in two hundred should be lost in this way where the surrounding parts are healthy at the time of operation.

Severe attacks of *iritis* and *iridocyclitis* are also set up by infection. Such cases seem to originate, so far as is known, in the remains of the lens tissue. Lens cortex has been shown experimentally to be the most favourable tissue in the eye for the growth of organisms. The result of *iritis* when the inflammation remains limited to the more anterior portions of the eye

is to leave a more or less dense screen, consisting of iris, capsule, and fibrinous exudation, behind which the vitreous may be more or less infiltrated. The contraction of the iritic exudation often leads to a greater or less obliteration of the coloboma, when an iridectomy has been performed, so that the pupil is drawn up towards the external wound. Occasionally an infected *iritis* after cataract extraction may lead to sympathetic mischief (*vide* "Sympathetic Ophthalmitis").

Lesser degrees of *iritis* are frequent. They do not cause any displacement of the pupil, though some synechiae form. They are probably not of an infected nature, but caused mostly by the irritation of remaining cortex.

*Glaucoma* may also occur as a complication of the healing process (*vide* "Secondary Glaucoma").

A serious accident which sometimes occurs, and which cannot be foreseen, is copious bleeding leading to detachment of the retina, which is then often protruded through the external wound. This may occur any time within the first twelve hours after operation. Most commonly it takes place almost immediately afterwards. The eye has then to be enucleated.

Only when the pupil is perfectly free from cortical remains is the sight on proper optical correction as good twenty-four hours after operation as it eventually becomes. As a rule, owing to gradual absorption of the matter left in the eye, vision slowly improves, and only reaches its maximum after, it may be, a good many weeks.

In by far the largest proportion of cases the best results, so far as the vision is concerned, are only to be got by a second operation, which consists of *needling* the capsule. This operation, though very safe when properly done, should be avoided whenever the vision is sufficiently good otherwise for all practical purposes. Not only is there some immediate risk attaching to it, some chance of either inflammation or glaucoma, but clinical evidence certainly appears to support the view that an eye in which the vitreous has been disturbed cannot be regarded as quite so safe as it would otherwise be. In all cases, therefore, the vitreous should be disturbed as little as possible. This is best attained by making the desired opening in the capsule with a single cutting needle or fine knife. The instrument should be entered through the corneo-scleral margin at a point which lies outside of the cicatrix of the original extraction wound. Its point should then be passed through the capsule at the opposite side of the pupil and the cutting edge directed backwards. Finally, the handle of the instrument is raised, causing the blade to sweep in the opposite direction and cut an opening in the capsule. This should be done in such a way, too, as to prevent the needle passing deeply into the

vitreous. In most cases where needling is done in this way it may be done in the course of a fortnight after the first operation. The capsule is then elastic, and readily retracts so as to leave a gaping opening where it has been cut. The contra-indications to early needling are any abnormal degree of reaction following the extraction and the presence of much soft free cortex.

In cases where owing to iritis the pupil has been closed and drawn up with the iris fibres put on the stretch, the opening is best made with a pair of small scissors constructed for the purpose (iridotomy scissors). An opening, which should not be too small, is made with a keratome in the corneo-scleral margin and the scissors introduced, with the sharp-pointed blade pushed behind the iris and the blunt one in front. As soon as the blades have passed over to the opposite side of the anterior chamber a snip is made. The cut, being at right angles to the stretched iris fibres, admits generally of sufficient retraction of these to maintain an opening. In the worst cases a bit of the opaque screen has to be cut out. In all such cases, however, the prognosis is less favourable.

The large opening made in the eye for the extraction of the crystalline lens causes, as might be expected, some alteration in corneal curvature. This *operative astigmatism* has to be taken into account in determining the optical correction afterwards required for near and distant vision. It is some time—generally a good many weeks—before the amount of the astigmatism has become constant. The progress of coarctation leads to its getting gradually less and less for some time. The final astigmatism varies in amount in different cases. This depends partly upon how the section has been made and how healing has taken place, but also upon the previous state of the eye as regards astigmatism. The operative astigmatism more frequently counteracts than supplements a previously existing astigmatism, as it is the horizontal meridian of the cornea which is most curved after operation. When the factor of pre-existing astigmatism is taken into account it will be found that for the same operator the acquired astigmatism is much the same whenever healing has taken a normal course.

One question remains to be considered, viz. How far in the case of slowly developing cataract is it necessary or advisable to wait until the cataract is ripe before operating? This question would no doubt be answered differently by different surgeons. The writer can only here offer such advice as his own experience leads him to consider sound. It has to be remembered that many unripe cataracts, especially in old people, can be extracted just as readily and as completely as can ripe cataracts. In many cases, however, a good deal more cortex remains in the eye. The cortex is often not only less

readily removed but, being transparent at the time of operation, cannot be recognised. As the time taken for the sufficient clearing of the pupil depends upon the absorption of remaining cortex, unripe extractions take on the average somewhat longer before useful vision is restored, while the risk of iritis of all kinds is proportionately increased. A much larger proportion, too, require the second operation of needling. Against these disadvantages have to be put those of having to remain for years without any useful degree of vision if one waits till a cataract is ripe.

An estimate of the relative importance of the *pros* and *cons* in this case can only be arrived at by personal experience. The following line of action is suggested here—

1. Do not hesitate to operate on cataract that is unripe, provided the patient has otherwise healthy eyes and adnexa and the vision is so far reduced as to interfere seriously with comfort, *e.g.* makes the reading of ordinary type or the following of the ordinary occupation impossible.

2. Do not operate on unripe cataract of one eye as long as the other eye retains sufficiently useful vision.

3. Wait till the cataract is ripe in all cases in which the risks of extraction from any cause are evidently greater than usual.

4. Do not perform preliminary iridectomy or any other operation for the artificial ripening of the cataract.

The practical result of following these rules is that one operates on a much larger proportion of unripe than of ripe cataract, but that when one has to regret the failure of the operation from serious subsequent complications it is mostly in cases in which there was not much to be lost at any rate.

**CATARACT GLASSES**—After extraction, the eye, if previously emmetropic, becomes necessarily hypermetropic. The glass which then corrects for a distance when placed about 15 mm in front of it is one of 10.0 or 11.0 dioptres. That is, in the condition of *aphakia* (absence of the crystalline lens) the previously emmetropic eye has to be provided with a glass lens of +10.0 to +11.0 in order to see distant objects distinctly. The retinal images then got of external objects besides being sharp are larger than they were previously in the proportion of about 3/2. If the eye were previously hypermetropic a stronger lens would be required for correction, *e.g.* if the degree of hypermetropia were 5.0 D, the glass after extraction would require to be +16.0 to +17.0. If the eye were previously myopic the correcting glass on the other hand would have to be weaker, *e.g.* if the previous degree of myopia were 5.0 D the lens after extraction would have to be +6.0 to +7.0, if 10.0 D, +3.0 to +4.0, if 15.0 D, +0.75 to +1.50, if 20.0 D, -1.50 to -1.0 approximately. In the last case, therefore, the operative

hypermetropia would not altogether counteract the previous myopia.

With the absence of accommodation necessarily following the removal of the crystalline lens (*vide* "Accommodation"), a stronger glass than that used for a distance is always required for reading. As a general rule the reading glass should be about 4.0 D stronger, therefore in the case of previously emmetropic eyes +14.0 to +15.0. But this depends upon the distance at which the individual reads. With good visual acuity he may prefer to use a weaker reading glass. Considerable alteration in focus is also got by sliding the spectacles up and down the nose, as their effect is stronger the further they are removed from the eye. In this way what is tantamount to a certain range of accommodation is practically obtained.

Cataract lenses should always be of glass, not of crystal (pebble). Glass does not transmit the actinic rays to the same extent, and these rays are hurtful to the eye.

Cataract lenses diminish the field of vision very materially. On this account they should be worn as large as possible, compatible with not too great weight. As a general rule the best vision is only obtained with spherocylindrical lenses, that is, lenses one surface of which is spherical and the other cylindrical. This is on account of the astigmatism already referred to. Spherocylindrical lenses are necessarily heavier than ordinary biconvex spherical lenses.

When only one eye has been operated upon and the other is of little use, the spectacles may conveniently be made reversible, i.e. the one side provided with the distant and the other with the reading glass.

Cataract glasses should not be worn as long as there is much cortex left to be absorbed. In any case it is inadvisable to use them sooner than a fortnight after extraction. When used soon they have often to be changed after some months owing to the change which takes place in the amount of operative astigmatism.

**Catarrh.**—Inflammation of a mucous membrane. See BRONCHITIS, BRONCHITIS, BRONCHITIS ("Catarrh sec"), HAY FEVER, INFLUENZA, MEASLES (*Analysis of Symptoms, Catarrh*), NOSE, ACUTE INFLAMMATION (*Acute Rhinitis or Coryza*), NOSE, CHRONIC INFLAMMATION, STOMACH AND DUODENUM, DISEASES (*Gastritis*).

**Catarrhe Sec (Laennec).**—Dry chronic bronchitis, with severe paroxysms of coughing. See BRONCHITIS (*Clinical Varieties*).

**Catarrhus Æstivus.** See HAY FEVER.

**Catarrhus Communis.** See NOSE, INFLAMMATION (*Coryza*).

**Catarrhus Epidemicus.** See INFLUENZA.

**Catarrhus Ferinus.** See WHOOPING-COUGH.

**Catastasis.**—Constitution, habit of body, restitution or the diminution in the severity of the symptoms of a disease (in contrast to paroxysm).

**Catatony.**—A psychosis, described by Kahlbaum, characterised by cramps, tetanoid rigidity, catalepsy, melancholia, and stupor, it resembles melancholia attonita and general paralysis of the insane. See KATATONIA, SLEEP, NORMAL AND MORBID (*Morbid Somnolence*).

**Catchment Area.**—That part of a river basin from which rain is collected, usually for the purposes of water-supply.

**Catechu.** See ASTRINGENTS, DIARRHŒA, PHARMACOLOGY, PRESCRIBING.—Pale catechu (obtained from *Uncaria Gambier*) is an astringent extract, which contains *catechu-tannic acid*, catechuic acid or catechin ( $C_{15}H_{20}O_8$ ), and pyrocatechin or catechol ( $C_6H_4(OH)_2$ ). It is powerfully astringent, and is given in doses of 5 to 15 gr., it is incompatible with gelatin, alkalies, and metallic salts. Its preparations are *Pulvis Catechu Compositus* (dose, 10 to 40 gr.), *Tinctura Catechu* (dose,  $\frac{1}{2}$  to 1 fl dr), and *Trochiscus Catechu*. The lozenge (containing 1 gr of catechu) is a favourite remedy for sore throats. *Black Catechu* (*Acacia Catechu*) is not official in the British Pharmacopœia.

**Caterpillar Rash.** See RUBELLA, RICKETTSIA (*Diagnosis from Caterpillar Rash*), DERMATITIS TRAUMATICA ET VENENATA (*Causal Agents, Animal*), MYIASIS (*Many Caterpillars*).

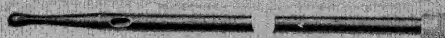
**Catgut.** See ASEPTIC TREATMENT OF WOUNDS (*Sterilisation*).

**Catharsis.**—The feebly caustic action of a substance.

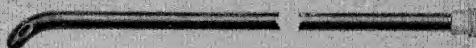
**Cathartics.**—Diastolic purgatives, such as calomel, jalap, aloes, scammony, colocynth, and croton oil, which produce a violent action of the bowels, usually attended with griping and sometimes followed by considerable prostration. Some of them (e.g. elaterium and scammony) cause a copious watery flow, and are called *hydragogue*. See also CONSTIPATION, PHARMACOLOGY, PRESCRIBING, and under the various drugs. *Catharsis* is the name given to the effect which cathartics produce (literally, *cleansing*).

**Cathelectrotonus.**—When a galvanic current is passed through a nerve that structure is found to be altered in condition at the poles, this altered state is called *electrotonus*, and the special condition of increased irritability found at

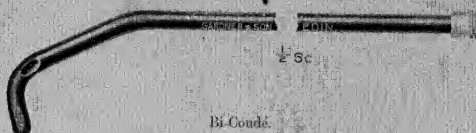




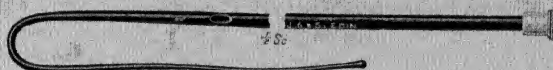
Solid-tipped Catheter.



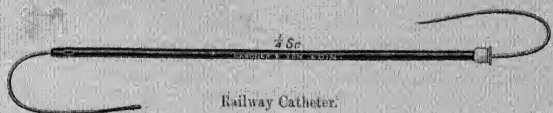
Coudé.



Bi-Coudé.



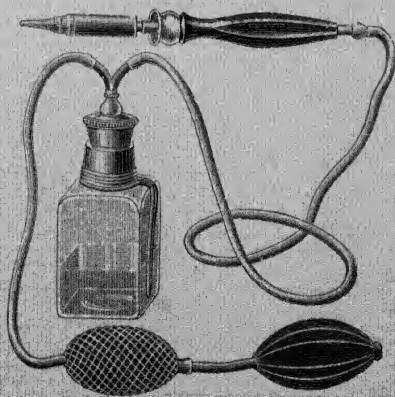
Whip Catheter.



Railway Catheter.



Corrigan's Button Cautey.



Paquelin's Thermo-Cautery.



useful when there is a dilatation at the bulb or a median enlargement of the prostate. Force is quite inadmissible, the instrument must find its own way. If the catheter is a rigid one the change in the direction of the urethra as it winds under the pubes through the triangular ligament must be borne in mind, and care must be taken to depress the handle sufficiently. False passages, when they do not begin in front of a stricture, always start from this point, and generally run upwards and backwards between the prostate and the bowel. If the finger is introduced into the rectum the least deviation can be detected at once. In cases of enlargement of the prostate an English gum-elastic, which has been kept for some time over-curved upon a stilet, sometimes succeeds when others do not, or Hey's well-known plan may be tried of passing the catheter down to the obstruction, withdrawing the stilet, and at the same time gently pressing the instrument onward. In stricture cases a catheter with a very long and fine whip-like end beyond the eye will sometimes find its way. Small metal catheters require the greatest care, as the points are very sharp and the walls of the urethra very tender. When the object is to draw off a large amount of residual urine, and it is essential to prevent any organisms being carried into the bladder, an instrument like Melchior's should be used. This consists of two tubes, one gliding inside the other. The outer is of metal, and is sufficiently long to reach the membranous portion. Its vesical end is closed with a film of rubber held in place by a metal cap. As soon as this is in position, a smaller flexible one is pushed down through it and through the rubber film directly into the prostatic urethra, so that the catheter which enters the bladder is never brought into contact with the meatus or the mucous membrane of the anterior portion of the urethra.

**PAIN AND SPASM**—The urethra is exceedingly sensitive, especially the deeper portion. The passage of an instrument through this is always accompanied by pain and sometimes by syncope. All instruments must be warmed. Under the mucous membrane is a stout layer of unstriated muscular fibre which often grips the catheter firmly, and prevents it for a time from being pushed on or drawn back, but the contraction always tires itself out in the course of a few minutes. A preliminary injection of a few drops of a 2 per cent solution of cocaine will prevent both pain and spasm.

The passage of a catheter, especially for the first time, is liable to be followed by consequences, some of which, such as syncope and shock, are referable to the *reflex action of the nervous system*, while others are due to the *introduction of septic organisms* into the deeper part of the urethra and the bladder.

**SYNCOPE**—Syncope is not uncommon, and may be serious if in an old man a large amount

of residual urine is suddenly allowed to stream out through a full-sized catheter while he is standing upright. Shock is rarely grave or general, but local shock, due to inhibition of some of the nerve centres in the lumbar portion of the spinal cord, is not at all uncommon. It may show itself by retention from inhibition of the centre which controls the act of micturition, or by hæmaturia from congestion of the kidneys due to temporary vasomotor paralysis, or even by suppression of urine. As the cause is a very transient one the effects nearly always subside without any active measures being necessary.

**SEPTIC POISONING**—The effects which are due to the introduction of septic organisms may be either local or general. The former include the inflammatory affections of the urethra, bladder, epididymis, and prostate, which are so often met with after careless catheterism, the latter comprise the different forms of what is miscalled catheter or urinary fever. The bacillus coli is the most common septic organism, but the staphylococcus aureus, citreus, and albus, the streptococcus pyogenes, and the bacillus ureæ liquefaciens are all of frequent occurrence, alone and together. They may enter the urinary passages through the blood-stream or the lymphatics, invading the walls first, or they may come down from the kidneys in the urine, or ascend upwards from the meatus (especially in the case of women), but when febrile attacks occur after the introduction of a catheter, the organisms which cause them are nearly always carried in on the surface or in the eye of the instrument. It is for this reason that sterilisation is so important.

The *simplest form of urinary fever* is a rigor or shivering fit, due to absorption through some abrasion (or perhaps even through the unbroken surface of the urethra) of the toxins which have been formed by septic organisms growing in the urethra. It begins as a rule, not when the catheter is passed, but some hours later, after the first act of micturition. All of a sudden the patient is seized with intense prostration and chilliness, which is described as being felt inside. The skin becomes rough and livid. The face is pinched. The eyes look sunken, and are surrounded by dusky rings. The respiration is hurried and shallow, and the pulse small and frequent. From the first moment, even when the teeth are chattering and the patient shivering, the temperature begins to rise, and it continues through the period of dry, burning heat which follows until sometimes it reaches 106° F. In ten minutes or half an hour, according to the severity of the attack, profuse perspiration sets in, the face becomes flushed, the involuntary muscular fibre in the skin relaxes, and there is a sensation of profound relief although the patient is utterly exhausted.

Rigors do not occur even after such operations as internal urethrotomy if care is taken to render

the instruments, the urine, and the urethra aseptic. They are more rare after operations upon old and dense cartilaginous strictures than after those upon recent ones, not because the germs are fewer, but because in the former absorption is not so easy. They occur, as a rule, not when the catheter is passed, but afterwards at the first micturition, because the passage of the urine down the urethra raises the pressure and forces into the circulation the toxins which have been formed in the interval. They seldom occur after external urethrotomy, because owing to the open wound there is no pressure. After lateral lithotomy they are very rare, even when the urine is septic, for the same reason. And the same thing explains what has been so often noted, that, while every attempt at dilating a stricture causes a rigor, free urethrotomy or rapid and complete stretching cures the patient at once without the least reaction.

There are, of course, accessory causes. Any slight chill after an operation upon the urinary organs, such as standing with bare feet upon cold oil-cloth, will precipitate a rigor, and patients who have been exposed to malaria are especially predisposed to such attacks. But these are not necessary, and there can be no doubt, from what has been learnt by experimenting upon animals, that the toxins are perfectly efficient by themselves, even though the amount which is driven into the circulation is infinitesimal.

The prognosis in this form of urinary toxæmia is good as a rule. In some, fortunately very rare, cases the collapse has been so severe that the patient has never rallied. This is recorded as having happened even after the passage of a catheter, but most of the cases have followed operations upon the kidney for septic pyelitis. The explanation is that the manipulation of the kidney during the operation forces into the open ends of some of the renal venules such a large amount of the toxins which have been lying stagnant in the pelvis that the nerve centres are simply overwhelmed. Even when there are two or three rigors in succession there is no great cause for alarm. It usually means the absorption of successive doses. But when the rigors continue after forty-eight hours, after the surface of the wound in the urethra has been glazed over, it becomes a question whether the organisms themselves have not entered into the blood-stream as well as the toxins, whether it is not a case of septic infection rather than of simple toxæmia.

True septic infection may, of course, occur after operations upon the urethra as after operations upon other parts of the body. There are no local symptoms, but the rigors recur again and again, generally irregularly, but sometimes with intervals as regular as in ague. The patient rapidly becomes weaker and thinner. Perhaps slight jaundice sets in. There may be an attack of diarrhoea with peculiarly offensive motions.

The pulse becomes more and more frequent, and at length death ensues from exhaustion.

*Another and equally common form of what is called urinary fever is more chronic and much more insidious.* The most typical examples are met with in cases of enlargement of the prostate at the beginning of catheter life, especially in those in whom there is a large amount of residual urine. A catheter is passed and the bladder partially emptied. This is repeated once or twice a day for several days. There is no rigor or other symptom of note. The urine is clear, but the specific gravity is low, and the amount unduly large. On the fourth or fifth day the bladder becomes irritable, and the urine is found to be a little turbid, so that after standing a grey deposit of pus settles at the bottom of the vessel. The total amount may increase until it reaches seven or eight pints a day, or it may diminish. The reaction is generally faintly acid, but it rarely fails to become alkaline after the first few days. There are often a few hyaline casts, and sometimes there is a little albumen. There is no rigor though the patient may complain of feeling chilly. The temperature only rises one or two degrees, but the pulse increases in frequency and diminishes in strength, the tongue becomes dry and brown, especially down the centre, the appetite is lost, there is a tendency to nausea, the mind becomes a little unsettled, and then muttering delirium begins at night, and the patient sinks into what is called a typhoid state. Post-mortem the bladder may be found to be large, with thin walls, or small and rigid, with thick ones, and perhaps sacculi projecting from them, but there is always evidence of recent acute cystitis. The ureters are dilated and full of offensive urine and pus. The pelvis of the kidneys is in the same condition. The mucous membrane is discoloured, with flakes of pus adhering here and there. The apices of the pyramids are eroded. The kidneys themselves are small and hard, and on section are seen to be studded with minute abscesses differing in shape and outline according to the anatomical arrangement at that particular spot. In a word, there is evidence everywhere of long-standing disease of the bladder and kidneys with, in addition, recent septic inflammation and suppuration. The symptoms are not due to shock caused by emptying the bladder. They are in part the result of chronic renal insufficiency, in part of septic poisoning. The fault is in the bladder not being emptied properly. A certain amount of urine, a liquid in which micro-organisms grow exceedingly well, is habitually left stagnant in its recesses. Day by day more and more organisms are introduced by the catheter, and at last septic cystitis and pyelonephritis result.

*Treatment* — Nothing special need be said about syncope or shock when they follow the passage of a catheter or any other operation

upon the urethra Retention of urine can often be prevented by a subcutaneous injection of strychnia given at the time of the operation If it follows in spite of this a catheter must be passed again, but not until it is clear that there will be no relief without Suppression must be treated by subcutaneous injections of digitalin, cupping over the loins, hot baths, and purgatives

So far as what is commonly called urinary fever is concerned, the main treatment is prevention All instruments should be sterilised beforehand, and only touched with hands that have been rendered thoroughly aseptic The patient's bowels should have been well opened, preferably by a purge containing calomel, and the urine should have been rendered as resistant as possible to living organisms by the administration of intestinal antiseptics for some days beforehand Salol and naphthalam I believe to be the two most efficacious, but the former should not be given in doses larger than three or four grains at a time, for fear of forming intestinal concretions The patient should remain quiet for some hours after the operation, even if it has been merely the passage of a catheter (unless he is accustomed to it), and great care must be taken to avoid anything that might cause a chill

The bladder should be emptied at the time of the operation, and micturition postponed for as long as the patient conveniently can There will then be less risk of absorption A hot hip bath just before the first act of micturition lessens the chance of a rigor, not only by its action upon the skin, but by the way in which it causes all the unstriated muscular fibre around the urethra to relax, and so lessens the pressure as the urine is driven down At the same time the patient may be given a cup of hot tea, with some brandy, opium, and quinine

It is probably impossible to render the mucous membrane of the urethra really aseptic, certainly if it is inflamed, but there is some advantage, I believe, in washing it out thoroughly before such an operation as internal urethrotomy, either with a saturated solution of boracic acid or with nitrate of silver, one in a thousand After a stricture has been divided in this manner I always inject on to the face of the wound twenty drops to half a drachm of a  $\frac{1}{2}$  per cent solution of nitrate of silver or of protargol, and leave it there The same thing should be done at the neck of the bladder in cases in which a large amount of residual urine is evacuated It certainly checks, if it does not absolutely prevent, the growth of septic organisms at the critical place and time

If a second rigor occurs after internal urethrotomy or any other operation upon the urethra, the injection should be repeated, and a catheter tied in so that the bladder may be

kept permanently empty for forty-eight hours. This renders further absorption almost impossible. It is not wise to leave the catheter longer than forty-eight hours, for fear of causing urethritis

If the bladder becomes irritable, or if any deposit of pus makes its appearance after the evacuation of residual urine, the same thing should be done, and in addition, if the state of the bladder will allow it, the bladder itself should be washed out twice a day with corrosive sublimate or nitrate of silver. If this does not succeed very soon, or if it is evident from the shape of the prostate, or the condition of the bladder, that it is not possible to keep the cavity empty by tying a catheter in, there should be no hesitation in adopting perineal drainage or even puncture through the prostate. If septic cystitis is allowed to continue, pyelitis and pyelonephritis will follow The most successful way of stopping inflammation of the bladder is to follow the plan adopted by nature so far as it can, and empty the bladder and keep it empty The danger lies in leaving a collection of urine, which has become converted by septic organisms into a virulent poison, stagnant in the post-prostatic pouch The wisest plan is to adopt adequate measures while there is yet time

Very little can be done in these cases towards restoring the aseptic character of the urine by means of drugs Unhappily, intestinal antiseptics have very little influence upon the urine They may make it less favourable as a nutrient medium for the growth of organisms, but the amount that reaches the urine through the kidneys is not sufficient to kill organisms which have already established themselves And it must not be forgotten that the majority of patients who are attacked by this form of urinary fever are already enfeebled by long-standing renal inadequacy, and that anything which tends to upset their digestion, or prevent their taking sufficient nourishment, is more likely to do them harm than good Quinine, boric acid, salol, and naphthalam are the drugs which I have found most useful, but the patient must not be forgotten while the complaint from which he is suffering is being treated

**Cathetometer.**—A catheter gauge for ascertaining the calibre of the instrument

**Cathode.**—The negative pole of a galvanic battery, the negative electrode

**Cattivo Male.** See PELLAGRA

**Cattle.** See ABATTOIRS, ANTHRAX, DAIRIES, FOOT AND MOUTH DISEASE, RHEUMATISM, CHRONIC (*Comparative Pathology*), TUBERCULOSIS, etc.—From the Public Health point of view, *cattle* includes bulls, cows, oxen, heifers, and calves, while the term *animal* is

applied to cattle, sheep, goats, all other ruminants, and swine. In Scotland, however, the former term includes sheep, goats, and swine.

**Cattle Plague.**—A markedly contagious disease, characterised by fever, catarrh (nose, eyes), cessation of rumination, constipation, etc.; rinderpest. See *INUNIA* (*Cattle Plague*).

**Cauda Equina.**—The bundle of leadsh of nerves arising from the spinal cord at the level of the second lumbar vertebra, and including the conus medullaris or terminal part of the cord itself. See *SPINAL CORD, MEDICAL, SPINAL CORD, SURGICAL*, etc.

**Caudate Nucleus.** See *BRAIN, PHYSIOLOGY, PHYSIOLOGY, NERVOUS SYSTEM (Cerebrum)*.

**Caul.**—If during labour the fetal membranes (amnion and chorion) or one of them (amnion) fail to rupture till after the head of the child is born, or if the rupture take place high up inside the uterus, the infant is born "with a caul," i.e. with the head enveloped in the membranes, the sillyhow or *caput galeatum*. See *LABOUR, FAULTS IN THE PASSENGER (Membranes, Toughness)*.

**Cauliflower.** See *INVALID FEEDING (Vegetables)*.

**Causalgia.**—Neuralgia, accompanied by a burning sensation. See *NERVES, NEURALGIA*.

**Causis.**—Cauterisation. See *ATMOCAUSIS, ZESTOCAUSIS*.

**Caustics.**—Substances which, when applied locally, kill the tissues with which they come in contact and set up inflammation in the surrounding parts, escharotics, instances are found in nitric and sulphuric acid, in caustic potash and caustic lime, in carbolic acid, and in arsenious acid (*q.v.*). Iron at a red heat also acts as a caustic, as does the galvano-cautery. See *CANQUOIN'S PASTE, CAUTERY, ESOPHAGUS (Inflammation), TOXICOLOGY (Corrosives)*, etc.

**Cauterets.** See *BALNEOLOGY (France, Sulphur), MINERAL WATERS (Sulphated)*.

**Cautery.** See also *CAUSTICS, GALVANIC CAUTERY; HÆMORRHAGE (Cauterisation)*.—There are three varieties of cautery: (1) The actual cautery, the application points being usually hatchet-shaped, globular, or flat, (2) thermor-Paquein cautery, the terminals being straight, curved, knife-like, pointed, or flat, and (3) galvano-cautery.

**HEAT AT WHICH EMPLOYED.**—This may be black, red, or white heat, and varies according to the object of its use.

**BLACK HEAT.**—The cautery is used at black

heat as a counter-irritant. This is useful in many chronic affections of bones, joints, and serous membranes, such as spinal meninges, pleura, bursa, tendon sheaths, in chronic conditions of spinal cord, nerves, especially intercostal neuralgia and sciatica. It is also of service in hysterical conditions, with localised painful areas, and in cases of malingering.

**RED HEAT.**—It may be used at a dull or a bright red heat. (1) At a dull red heat the cautery is used as a severer form of counter-irritant in the conditions mentioned previously, and also as a hæmostatic in cases of hæmorrhage where a ligature cannot be applied, e.g. bleeding from bone or tooth sockets, or oozing from a large surface, or in hæmophilic subjects. Occasionally it is required when tracheotomy has to be performed through a very vascular growth.

(2) At a bright red heat it is not applicable as a hæmostatic, the eschar being too weak. It is useful, however, as a severe counter-irritant, and also for touching fistulous openings, e.g. urethra, rectum, umbilicus, salivary ducts, with a view to encouraging them to close. Phagedenic sores and the surface of poisoned wounds are often much benefited by its use. It is also employed for the removal of urethral caruncles, prolapse of the rectum, growing capillary nevus, aneurysm, and also ignipuncture of hypertrophied tonsils. The employment of the cautery for the removal of tumours is now abandoned.

**WHITE HEAT.**—This is applicable in all the previously mentioned conditions, except where the hæmostatic action is desired. It is the most useful heat to employ where tissue has to be punctured to some depth, as in ignipuncture of tuberculous glands, as it retains the heat for a longer time. Negreth has recommended its use in hypertrophy of the prostate, the gland being punctured from the rectum.

**TECHNIQUE.**—1. *Actual Cautery.*—When black heat is to be used the button cautery, or Corrigan's button, is the form employed, a latch key making a good substitute. When the cautery has been heated in the flame of a spirit-lamp until the metal shaft close to the wooden handle becomes so hot that it only allows of momentary painless touching with the finger, it should be rapidly tapped over the painful areas. For red or white heat the cautery is placed in a fire, and on removal is applied whenever its temperature has reached the desired limit. The hatchet-shaped instrument is used for linear cauterisation, the parallel lines being drawn at a distance of about one inch apart. If the flat form be used it is drawn over the surface at the rate of about one inch a second. For hæmorrhage the bleeding surface is steadily seared by rubbing the cautery over it, otherwise the instrument may adhere, and in its removal separate the eschar, and the hæmorrhage may

be again started. If the bleeding point is in bone or the socket of the tooth, a knitting-needle at red heat bored against the point acts well.

2 *Sherrow Cautery* or *Paquelin's*.—Before use the instrument should always be carefully tested, special attention being paid to the following points.—The benzoline reservoir should be only half filled, and the benzoline should be fresh, as it rapidly loses the active and more volatile portions. The blade should be heated red-hot in a spirit-lamp, and then the vapour pumped in. Should it not work, most probably the benzoline is at fault, and the heat of the hand over the reservoir may be necessary to distil over some active vapour.

It is used in the same way as the actual cautery, but has one great advantage, that is, when it becomes adherent to the tissues the temperature can be raised and the cautery removed without detaching the eschar. After use the blade should be allowed to cool and then be dried. In ignipuncture for a painful spine, the skewer-shaped cautery is pushed through the skin over the lateral soft tissues, and inserted about  $\frac{1}{4}$  to  $\frac{1}{2}$  of an inch. The punctures are made about one inch apart.

The Dechery automatic cautery is another variety of the thermo-cautery. Here ether is used instead of benzoline, half an ounce being sufficient for forty minutes' incandescence. This is a very convenient and portable form of instrument.

**PRACTICAL POINTS IN THE APPLICATION.**—(a) When the cautery is employed at red or white heat a *general anæsthetic* should be administered, and if ether be used great care must be adopted lest the inflammable vapour become ignited by the cautery.

(b) If the *constitution of the patient* is enfeebled by *anæmia*, Bright's disease, diabetes, melancholia, or other cause, the use of the cautery at red or white heat should be avoided, the reparative power of the tissues being much diminished. For the same reason it ought not to be used, if from disease or from injury the trophic power of a part is impaired.

(c) *Area of Application*.—All hairy parts should be shaved, and the surface rendered aseptic. All open surfaces, as sores, fistulae, should be dried, and any blood-clots present removed. If the cautery is to be used for localised tender spots they should be marked before commencing the application. Soft parts with thick skin are the most suitable surfaces for application.

When superficial, all bones, nerves, tendons should be avoided, also the trachea and urethra. In brunettes, the neck, arms, and face are better avoided, as permanent pigmentation may result.

(d) **THE LOCAL RESULTS.**—The effect at first is very painful, especially when red and white

heat have been applied. The application of the cautery produces burns of various degrees at the point of application, and "radiation burns" in the immediate neighbourhood. The eschar following burns of the third and fourth degree may take several days to separate. When the cautery has been employed as a hæmostatic, the separation of such eschars are apt to be followed by hæmorrhage. After ignipuncture the healing may be very slow.

**AFTER-TREATMENT.**—If there be much pain anodynes internally may be given, except in hysterical patients and inhalerers.

In burns of the first and second degree a weak antiseptic ointment spread on lint is all that is required (see "Burns").

In severer forms of burn cold boracic compresses relieve the pain. If delay in the healing is desired, ung. sabine two parts to vaseline one or two parts should be rubbed in once a day, and if the pain so induced be very great 2 per cent to 10 per cent cocaine may be added. The eschar being aseptic does not interfere with the healing of a wound by primary union. The disagreeable odour of burning flesh may be partially concealed by the fumes obtained from placing the red-hot cautery in some ground coffee.

**Cavernitis.**—Inflammation of the erectile tissues (*corpora cavernosa*) of the penis. See PENIS, SURGICAL AFFECTIONS OF THE (*Inflammatory Affections*).

**Cavernous Breathing.** See LUNG, TUBERCULOSIS (*Physical Signs of Vesicular Formation*).

**Cavernous Sinus.** See BRAIN, PHYSIOLOGY (*Venous Circulation*).

**Cavities.**—Hollow spaces in the body, open or closed, e.g. abdominal, amniotic, buccal, ciliary, cranial, nasal, peritoneal, pleural, tympanic, and uterine cavities, also pathological formations, as in the lungs in phthisis (see LUNG, TUBERCULOSIS OF).

**Cayor Fly.**—An insect found in Africa, the larvæ of which apparently cause boils.

**Cebocephalus.**—A rare teratological type, closely related to cyclopia, in which the two orbits are approximated (but not fused), the nose deformed, and the whole face diminished in size. The expression of the face is ape-like (*ἄψος*, ape, *κεφαλή*, head). The brain shows the same malformations as in cyclopia.

**Celerina.**—A preparation, said to contain celery, coca, kola, viburnum, and aromatics, which is recommended as a nerve tonic and antispasmodic in cases of neurasthenia, neuralgia, dysmenorrhœa, etc.

**Celery.** See INVALID FEEDING (*Vegetables*).

**Celibacy.**—The unmarried state; suicide more common among celibates than among the married. See SUICIDE

**Cellars.**—Cellars are prohibited as dwellings by the Public Health Act (England and Wales) of 1875, with certain exceptions carefully specified in the Act. The Public Health (Scotland) Act of 1897 contains similar restrictions

**Cellotropin.**—One of Merck's preparations, obtained by the action of benzoyl chloride upon arbutin, of which it is the monobenzoylester, it is used (in doses of 5 to 8 grains) to protect the system from infection (by the formation of alexines)

**Cells.** See PHYSIOLOGY, THE CELL, PHYSIOLOGY, THE TISSUES (*Ethelium, Connective Tissue, Muscle, Nerve*)

**Cellulitis.**—Inflammation of the areolar tissue See ANEURYSM (*Complications, Cellulitis and Suppuration*), AXILLA, DISEASES (*Acute Cellulitis*), BURSE, INJURIES AND DISEASES (*Burns, Acute*), CHEST-WALL, AFFECTIONS (*Acute Cellulitis and Abscess*), MENSTRUATION AND DISORDERS (*Dysmenorrhœa from Pelvic Cellulitis*), MUMPS (*Local Affections, Cellulitis of Floor of Mouth*), NECK, REGION OF (*Inflammatory Affections*), ORBIT, DISEASES OF (*Orbital Cellulitis or Phlegmon*), PELVIS, DISEASES OF THE CELLULAR TISSUE (*Pelvic Cellulitis*), PELVIS, HEMATOCELE AND HEMATOMA, STERILITY (*Causæ*), TEETH (*Alveolar Abscess*), UTERUS, DISPLACEMENTS OF, UTERUS, NON-MALIGNANT TUMOURS (*Diagnosis*)

**Celluloid.**—An artificial substance consisting chiefly of the lower nitrates of cellulose in camphor, and used as a substitute for ivory, bone, etc., its great inflammability and consequent danger have been diminished by the addition of sodium and ammonium phosphates, etc., xylolite

**Cellulose.**—The chief component of vegetable structures, a carbohydrate of unascertained constitution ( $C_6H_{10}O_5$ )<sub>n</sub>, cotton-wool is almost entirely cellulose, nitric and sulphuric acids act upon it to form gun-cotton and celluloid (q v)

**Celosomus.**—A teratological type in which there is ventration of both the abdominal and thoracic organs through an opening (lateral or median) affecting the walls of both cavities (thorax and abdomen), the sternum is defective. It is derived from *κύλη*, a hernia, and *σῶμα*, body

**Cement.** See PHYSIOLOGY, TISSUES, TEETH (*Anatomy*)

**Cementoma.**—A fibrous odontome

which has ossified into a hard, cement-like tissue. See TUMOURS (*Odontomata, Cementoma*).

**Cemeteries.** See BURIAL-PLACES, CREMATION.

**Cenæsthesia.**—The vague consciousness of existence which exists apart from the special senses and is due to general impressions of bodily conditions, the name is derived from *κένωσις*, common, and *αἰσθησις*, sensibility, another spelling is *Cenæsthesia*

**Census.**—The decennial enumeration of the population of Great Britain (last was in 1901), in order to obtain a basis of facts for the science of vital statistics and for other reasons. The mean population is that at the centre of the year, and since the census is taken at the end of the first quarter, it has to be corrected for the three months which follow, to give the true mean population of that year. The census paper has to be filled up by the householder giving name, age, sex, occupation, birthplace, relationship to head of the house, married or single state, infirmities, etc., of the various people who slept in the house on the census night. It would probably be well to have the census more frequently taken, in order that the birth-rate and the death-rate, etc., should be more accurate

**Centimetre.** See METRIC SYSTEM

**Centipedes.** See NOSE, FOREIGN BODIES, ETC (*Parasites, Entomozoa*)

**Centres.** See BRAIN, PHYSIOLOGY, PHYSIOLOGY, THE SENSES, PHYSIOLOGY, NERVOUS SYSTEM, PHYSIOLOGY, CIRCULATION (*Varomotor Centres*), PHYSIOLOGY, RESPIRATION, PHYSIOLOGY, DIGESTION (*Vomiting Centre*), etc

**Centrifuge.**—A rotatory apparatus by means of which centrifugal force is used to separate solids from a liquid, e g milk or urine

**Centrosome.**—A sphenical body, sometimes two, found in the protoplasm of many cells, from which rays diverge, they play a part in mitotic division of the cell. See PHYSIOLOGY, THE CELL

**Cephalalgia.**—Headache, of various kinds, such as catarrhal, congestive, dyspeptic, epileptic, febrile, hysterical, menstrual, muscular, neuralgic, ocular, periodic, rheumatic, syphilitic, etc. See under the various diseases

**Cephalhæmatoma.**—A blood effusion (usually in the new-born infant) between the pericranium and one (or two) of the underlying cranial bones, to this form of tumour the term should be restricted, although it is often loosely applied to the caput succedaneum and to an effusion of blood inside the cranium (the so-called internal cephalhæmatoma). See





Cephalhematoma.



Cephalocele.



Cephalocele.



**BRAIN, SURGERY OF** (*Cephalocele, Diagnosis*); **HEAD** (*Cephalhæmatoma*), **NEW-BORN INFANT** (*Cephalhæmatoma Neonatorum*)

**Cephalic.**—Belonging or relating to the head, as in such expressions as cephalic index, cephalic lie (head presentation), cephalic tetanus, cephalic version, etc. See ANTHROPOLOGY, LABOUR, DIAGNOSIS and MECHANISM (*Presentations of the Vertex*), LABOUR, OPERATIONS (*Version*), TETANUS (*Clinical Features, Cephalic Type*)

**Cephaline.**—A substance derived from the brain, related to lecithine.

**Cephalitis.** See BRAIN, INFLAMMATION (*Encephalitis*)

**Cephalo.**—Cephalo-, occurring in many compound words, means relating to the head. It is used in many anatomical terms (*e.g.* cephalo-nasal, cephalo-humeral), and in such words as *cephalocentesis* (puncturing the head, as in hydrocephalus), *cephalograph* (an instrument for indicating cranial contours), *cephalomelus* (a teratological type in which a limb appears to spring from the head), and *cephalopyosis* (cerebral abscess), as well as in several names specially dealt with below

**Cephalocele.**—The swelling (skin-covered) formed by the protrusion of part of the cranial contents through a more or less rounded opening in the cranium, and it may be congenital or acquired. See BRAIN, SURGERY OF (*Cephalocele*), TERATOLOGY

**Cephalodynia.**—Headache, usually of a rheumatic and muscular type

**Cephalometer.**—An instrument, usually a pair of callipers, for taking the measurements of the head, a modified apparatus has been invented for gaining an idea of the diameters of the fetal head before birth (fetal cephalometry), a craniometer

**Cephalopagus.**—A double monster, or twins united to each other in the region of the head

**Cephalopline.**—An extract of brain substance.

**Cephalothoracopagus.**—A double monster, or twins fused together in the regions of the head and thorax as far as the level of the umbilicus.

**Cephalotomy.**—An obstetric operation (for diminishing the size of the child's head) proposed to be carried out by saw-forceps or wire-écraseur, obsolete. See LABOUR, OPERATIONS (*Embryotomy*).

**Cephalotripsy.**—An obstetric opera-

tion in which the foetal head is crushed by a pair of strong forceps (*the cephalotribe*) capable of being approximated by a screw at the handles. See LABOUR, OPERATIONS (*Embryotomy*), LABOUR, PROLONGED (*Contracted Pelvis, Treatment*)

**Cera.**—Wax. Two forms are employed (as a basis for ointments and plasters) in the British Pharmacopœia, *Cera Flava* (yellow beeswax) and *Cera Alba* (white beeswax), the latter being obtained from the former by bleaching. Cera consists chiefly of *cerotic acid* ( $C_{27}H_{54}O_2$ ) and *myristyl* (or *myricyl*) *palmitate* or *myricin*, with a small quantity of *cerotene*, and with paraffin and *ceresine* as impurities

**Cerasin.**—An insoluble gum from the cherry, peach, or plum tree, it resembles bassorin, and is (chemically) calcium metagummate

**Cerastes.**—The "Horned Viper" of India. See SNAKE-BITES AND POISONOUS FISHES

**Cerates.**—The cerates (*cerata*) are ointments containing wax, and they are official in the U.S. Pharmacopœia. Goulard's cerate is now represented in a modified form (in the B.P.) by the *Unguentum Glycerini Plumbi Subacetatis*. See PRESCRIBING

**Cercomonas.** See PARASITES (*Protozoa, Flagellate*)

**Cereals.**—Edible grains, such as wheat, oats, barley, and rye. See DIET (*Vegetable Food*), PHYSIOLOGY, FOOD AND DIGESTION (*Vegetable Food-Stuff*)

**Cerebellum.** See BRAIN, CEREBELLUM, AFFECTIONS OF. See also BRAIN, PHYSIOLOGY, BRAIN, SURGERY (*Abcess*); HYDROCEPHALUS, LUNG, TUBERCULOSIS (*Complications, Nervous System*), MENTAL DEFICIENCY (*Atrophic Conditions*), PHYSIOLOGY, NERVOUS SYSTEM (*Cerebellum*), TABES DORSALIS (*Diagnosis*), VERTIGO

**Cerebration.**—"The assemblage of the cerebral actions consecutive to a perception" (Lewes). Unconscious cerebration or latent thought (Hamilton) is a cerebral or mental action occurring during sleep or during the time when the attention is otherwise occupied

**Cerebrin.**—A substance obtained from brain tissue (by boiling first with baryta solution and then with alcohol) said to have the formula of  $C_{17}H_{33}NO_3$

**Cerebritis.** See BRAIN, ATROPHY, INFLAMMATIONS (*Encephalitis*)

**Cerebrosis.**—Inflammation or irritation of the brain, mania

**Cerebro-Spinal Fever.** See MENINGITIS, EPIDEMIC CEREBRO-SPINAL

**Cerebrum.** See BRAIN, PHYSIOLOGY, BRAIN, AFFECTIONS OF BLOOD-VESSELS, BRAIN, TUMOURS, BRAIN, ATROPHY, HYPERTROPHY, CYSTS, INFLAMMATIONS, BRAIN, SURGERY OF See also UNDER ALCOHOLISM, ARTERIES, DISEASES OF; ATHEROSIS, BED-SORES, FLUIDS, EXAMINATION (*Cerebro-Spinal*), GOUT (*Irregular, Nervous System*), HEART, MYOCARDIUM AND ENDOCARDIUM (*Symptomatology, Embolic Process*), HYDROCEPHALUS, INSANITY, PATHOLOGY OF, LYMPHATIC SYSTEM, PHYSIOLOGY (*Central Nervous System*), MENINGES OF THE CEREBRUM, MENINGITIS, TUBERCULAR, MENINGITIS, EPIDEMIC, MENTAL DEFICIENCY, NOSE, ACCESSORY SINUSES, INFLAMMATION (*Intra-Urinal Complications*), NOSE (*Cerebro-Spinal Rhinorrhoea*), PARALYSIS, PHYSIOLOGY, CENTRAL NERVOUS SYSTEM, PNEUMONIA (*Complications*), RHEUMATISM (*Nervous Affections*), SPINE, SURGICAL (*Lumbar Puncture*), SYPHILIS, TABES DORSALES, TEMPERATURE (*Nervous Origin*), TETANUS, TETANY, UNCONSCIOUSNESS

**Ceresole Reale.** See BALNEOLOGY (*Italy, Chalybeate and Arsenical*)

**Cerium.**—The ovalate (*Ceru Orata*) is official, and is given in doses of from 2 to 10 grains, especially in the vomiting of pregnancy, it is insoluble in water. See PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Digestion*)

**"Cerolin."**—A proprietary preparation, called in England *cerdin*, said to contain the active principle of beer yeast, and to do good in boils and acne, etc., it is given in pill form (dose, 1½ grain)

**Cerotic Acid.**—An oxidation product of paraffin, produced by prolonged boiling with sulphuric acid and solution of bichromate of potash, its formula is  $C_{27}H_{54}O_2$

**Certification.** See INSANITY, TREATMENT, LUNACY, MEDICAL PRACTITIONER, MEDICINE, FORENSIC (*Certification of Deaths*).

**Cerumen.**—The secretion of the external auditory meatus and of its ceruminous glands, it contains potash, oil, stearin, a reddish pigment, etc., and it prevents the easy passage of insects into the ear, ear-wax. See EAR, EXTERNAL, DISEASES OF (*Abnormal States of the Cerumen*). Excess of the cerumen (and its removal) is called *cerumenosis*

**Cervical.**—Belonging to the neck, e.g. to the vertebral column in the region of the neck, to structures in the same neighbourhood, or to the neck (*cervix*) of the uterus. In compound words it appears as *cervico*, e.g. *cervico-brachial*, *cervico-bregmatic*, *cervico-vaginal*, etc. See ACROMEGALY (*Cervical Kyphosis*), BRACHIAL PLEXUS (*Surgical Affections of*), LYMPHATIC SYSTEM, PHYSIOLOGY AND PATHOLOGY, NERVES, NEURALGIA (*Cervico-Occipital*), SYRINGOMYELIA

(*Paralysis of Cervical Sympathetic*); UTERUS, INFLAMMATIONS (*Cervical Catarrh*)

**Cervix.**—The neck-like part of any organ or structure, e.g. *cervix uteri* (*q.v. infra*), *cervix femoris*, *cervix vesicis*

**Cervix Uteri.** See GENERATION, FEMALE ORGANS OF (*Uterus*). See also ABORTION (*Treatment, Restraint of Hemorrhage*), CHOREA (*Chorea Gravidarum, Treatment*), GONORRHOICAL INFECTION (*Endocervicitis*), GYNECOLOGY, DIAGNOSIS IN, LABOUR, PHYSIOLOGY OF, LABOUR, STAGES AND DURATION, LABOUR, MANAGEMENT, LABOUR, PRECIPITATE AND PROLONGED (*Faults in the Soft Passages*), LABOUR, INJURIES TO THE GENERATIVE ORGANS, LABOUR, OPERATIONS, MENSTRUATION AND ITS DISORDERS (*Stenosis of the Cervix, etc.*), PELVIS, PERINEUM AND PELVIC FLOOR (*Prolapse*), PREGNANCY, PHYSIOLOGY, PREGNANCY, AFFECTS AND COMPLICATIONS (*Vomiting*), PREGNANCY, HEMORRHAGE (*Accidental, Placenta Previa, Plugging*), PUERPERIUM, PHYSIOLOGY, PUERPERIUM, PATHOLOGY (*Puerperal Infection, Mucous Plug in Cervix*), STERILITY (*Causes, Cervical Atresia*), SYPHILIS (*Acquired*), UTERUS, MALFORMATIONS, UTERUS, DISPLACEMENTS, UTERUS, INFLAMMATIONS, UTERUS, NON-MALIGNANT TUMOURS, UTERUS, MALIGNANT TUMOURS

**Ceryl.**—A hydrocarbon radicle ( $C_{27}H_{56}$ ), *ceryl alcohol* ( $C_{27}H_{55}OH$ ) is obtained from Chinese wax (*ceryl cerotate*,  $C_{24}H_{48}O_2$ ) by saponifying it, *cerylene* ( $C_{27}H_{54}$ ) is got (along with cetotic acid) by the distillation of the same substance (Chinese wax)

**Cesspools.** See SEWAGE AND DRAINAGE (*Cesspools*), TYPHOID FEVER (*Etiology, Sewage Emanations*)

**Cestodes (Cestolea).** See PARASITES (*Cestodes*)

**Cetaceum.**—Cetaceum or Spermaceti is a solid crystalline fat, obtained (mixed with oil) from the head of the *Physeter macrocephalus* or sperm whale, it consists of cetyl palmitate or cetine ( $C_{18}H_{38}C_{16}H_{32}O_2$ ), by saponifying it, cetyl alcohol or ethal ( $C_{18}H_{37}OH$ ) is got. Cetaceum is insoluble in water, but soluble in ether, chloroform, or alcohol (boiling). The official preparation is Unguentum Cetacei, and it is used in the making of ointments. See PRESCRIBING

**Cevadilla.** See VERATRINE

**Ceylon Sore Mouth.** See SPUR

**Chain.** See EHRLICH, IMMUNITY, etc

**Chalazion.** See EYELIDS, AFFECTIONS OF (*Glands of the Lids*).—A tumour developed in the eyelid due to inflammation of one or more Meibomian glands from blocking of the

ducts and retention of secretion, the contents in some cases become chalky (*chalazion terreum*), Meibomian cyst

**Chalcosis.**—Pneumonokoniosis due to the inhalation of silicious particles. See LUNGS, PNEUMOKONIOSIS

**Chalk.** See CALCIUM (*Calcium Carbonate*), GOUT (*Morbid Anatomy*)

**Challes.** See BAINEOLOGY (*France, Thermal, Sulphur*), MINERAL WATERS (*Sulphated*)

**Chalybeate Waters.**—Mineral waters containing iron, useful in anemia, chlorosis, debility, etc. See CHLOROSIS (*Treatment*), MINERAL WATERS (*Chalybeate*)

**Chamæcephaly.**—The shape of head in which the vertical index is less than 70.1° (or 75°), a low, flat head, due to marked synostosis of the temporal bones with the parietals, platycephaly. Similarly, *chamæprosope* is the term applied to a face with a small zygomatic facial index, a low face

**Chamomile.** See ANTHEMIDIS FLORES, PHARMACOLOGY, PRESCRIBING

**Champetier de Ribes' Bag.** See LABOUR, OPERATIONS (*Induction of Premature Labour*), PRUGNANCY, HÆMORRHAGE (*Unavoidable Hemorrhage, Treatment*)

**Champignon.** See TOXICOLOGY (*Fungi*)

**Chancery.** See LUNACY (*Chancery Lunatics in England*)

**Chancre.**—The initial lesion (or "hard sore") of syphilis, especially when in its classical form (as described by Hunter), the name is sometimes given also to the simple or non-infecting venereal sore. See SYPHILIS (*Modes of Infection, Acquired, etc.*), HERPES (*Herpes Genitalis*), VENEREAL DISEASE

**Chancroid.**—The simple, non-infecting venereal sore, the simple chancre, the soft sore. See PENIS, SURGICAL AFFECTIONS (*Herpes Progenitalis*), SKIN, BACTERIOLOGY (*Chancroid, Streptococcus of Ducrey*), VENEREAL DISEASE (*Differential Diagnosis*)

**Change of Life.** See MENOPAUSE, MENSTRUATION AND ITS DISORDERS (*Menopause*)

**Chaps.**—Cracks or fissures of the skin (e.g. on the hands in cold weather, or round the nipples during lactation), known also as *rimor* or *rhagades*, they are to be treated by careful drying after washing, byunction of glycerine or hazeline cream at bedtime. For cracked nipples see PUERPERIUM, PATHOLOGY (*Sore Nipples*)

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**Charas.**—A resin obtained from the leaves of Indian hemp, *churrus*. See CANNABIS INDICA

**Charbon.** See ANTHRAX, BOILS AND CARBUNCLE

**Charcoal.** See CARBO.

**Charcot's Disease.** See JOINTS, DISEASES OF (*associated with locomotor ataxia*), ANKLE-JOINT, DISEASES OF (*Tubercle Arthropathy*), HIP-JOINT, DISEASES OF (*Diagnosis, Charcot's Disease*), HIP-JOINT, DISEASES OF (*Neuro-Arthropathies*), KNEE-JOINT, DISEASES OF (*Neuro-Arthropathies*), OSTEO-ARTHIROPATHIES (*Charcot's Joint*), TARSUS DORSALIS (*Tubo-Arthropathies*)

**Charcot-Leyden Crystals.**—Octahedral crystals found in the sputum in asthma ("asthma crystals"), and in the blood in leukaemia. See BLOOD (*Examination, Special, Micro-Chemical*)

**Charpie.**—Old linen, unravelled, used for surgical dressings

**Chart.**—A printed form ruled with spaces for exhibiting in a graphic way rises and falls of temperatures, changes in pulse-rate, etc. See TEMPERATURE

**Charta.**—A paper covered or imbued with a medicinal substance, and used as a plaster. The *charta sinapis* (mustard leaf) is official. See PRESCRIBING, SINAPIS, etc

**Chartula.**—A small paper folded to contain a medicine in the form of a powder, rice paper may be used

**Chasmus.**—Yawning. See PHYSIOLOGY, RESPIRATION (*Special Respiratory Movements*), YAWNING

**Chatel-Guyon.** See BAINEOLOGY (*France, Minuted Water*), MINERAL WATERS (*Minuted Saline*)

**Chaudfontaine.** See BAINEOLOGY (*Holland and Belgium*)

**Chaulmoogra Oil.**—*Oleum Gynocardia* or Chaulmoogra oil is not official in the British Pharmacopœia, but is so in the Indian and Colonial Addendum of 1900. It is expressed from the seeds of *Gynocardia odorata* or of *Gynocardia Prunus*, it contains chiefly gynocardic acid and glyceride, and its dose (given in capsules) is 5 to 20 m. In the Indian Addendum there is also an official *Unguentum Gynocardia*. It has been much used, internally and externally, in leprosy. See LEPROSY, (*Treatment*).

**Cheek, Fissure of.** See also TERA-TOLOGY (*Face*)—*Definition*—A malformation of

the lateral portion of the face, consisting in a cleft affecting the soft parts, or more rarely invading also the bones, arising during the embryonic period of antenatal life, and caused by delayed or irregular closure of the fissures between the fronto-nasal and superior maxillary and the mandibular processes.

*Varieties*.—From the anatomical as well as from the pathological and embryological stand-points fissures of the cheek may be divided into three kinds (1) fronto-maxillary, (2) commissural, and (3) anomalous.

The *fronto-maxillary*, ascending, oblique, or naso-genal fissure begins at a point in the upper lip just outside the common site of hare-lip, and passes upwards outside the nostril towards the inner angle of the orbit, it may there produce coloboma of the lower eyelid, and it may, in exceptional cases, extend to the outer angle of the orbit and even on to the temple. It is unilateral or bilateral, simple or complicated, it varies in width from a narrow cleft to a gaping opening, it usually has a red floor and red irregular margins, but it may at one or more places present a cicatricial appearance (antenatal partial cure?). When the underlying skeleton is also fissured the line of cleavage does not exactly follow the suture uniting the upper maxilla to the bones of the nose, generally the intermaxilla carrying the incisor teeth is internal to the fissure, but occasionally a premaxillary is found external to it, a peculiarity not yet satisfactorily explained. The complications are the presence of other facial fissures (hare-lip, commissural cleft of the same or of the opposite side), deformity of the eye or cyclids, cerebrial hernias and hydrocephalus, and the more distinctly teratological conditions of anencephaly, exencephaly, and adhesion of the placenta or amniotic membrane to the face or brain. It is very rare in animals (mammals).

The *commissural*, intermaxillary, or transverse fissure of the cheek (maciostomia, hiatus buccalis congenitus, commissural hare-lip, or coloboma of the cheek) differs in direction from the fronto-maxillary variety, it passes from the buccal commissure backwards, with an inclination upwards towards the masseter, and even when it proceeds further it rarely invades either the orbit or the external ear. It may be unilateral or bilateral, simple or complicated, and of great or little extent. When it is bilateral and extensive the mouth is converted into an opening reaching from one ear to the other. In the less marked degree the defect lays bare one or two molar teeth at the angle of the mouth. The opening of Stenon's duct can be seen in the upper margin of the cleft. The zygomatic arch may be cleft as well as the soft parts, and the upper maxilla may be small or deformed. Although it may occur as a solitary malformation, it is more common to find it

associated with such anomalies as pre-auricular appendages (in eighty-seven cases of pre-auricular appendage collected from literature sixteen showed also commissural fissure, *v. Teratology*, n p 31, 1895), microcephaly, or hemiatrophy of face or cranium, fronto-maxillary fissure, deformity of the external ear, cleft palate, macroglossia, and exencephaly. In animals (lamb, calf, pig) it is, on account of its extent, a grave condition, for the mouth may communicate with the tympanic cavity, it is called schistocephalus megalostomus.

*Anomalous* fissures of the cheek which do not follow the lines of the natural embryonic clefts, but which pass, for instance, from the angle of the mouth to the inner end of the orbit, or obliquely across the face from one side to the other, have been put on record. It is probable that they are usually the result of an amniotic adhesion, the other two varieties being due to simple pressure from an imperfectly developed amnion.

*Symptomatology*.—In addition to the obvious deformity caused by fissures of the cheek the symptoms of the commissural variety in particular consist in imperfect closure of the mouth, dribbling of saliva, and difficulty in mastication and articulation. Infants exhibiting this deformity are often small, weakly, and poorly nourished, and may be defective in intelligence.

*Treatment* has almost invariably consisted in the closure of the fissure by paring and uniting by sutures its margin. Generally the operation has been quite successful.

**Cheese.** See DIET (*Milk and its Products*), PHYSIOLOGY, FOOD AND DIGESTION (*Milk, Cheese*), TOXICOLOGY (*Animal Foods, Cheese*), TUBERCULOSIS (*Tubercle and Butter Bacillus*).

**Chello.**—In compound words *cheilo-* (Gr *cheilos*, the lip) means relating to the lips, among such words may be named *cheiloplasty* (restoration of the lips to their normal form and continuity by operation, *vide* PALATE, HARE-LIP), *cheilostichia* (hare-lip), *cheiloscanceroma* (cancer of the lip), etc.

**Chelr.**—In compound words *cheir-* (Gr *cheir*, the hand) means relating to the hand, instances are found in *cheiromegaly* (giant growth of the hand), *cheiragra* (gout in the hand), *cheiroparalysis* (cramp in the hand), *cheiurgia* (surgery), etc.

**Chelropompholyx.** See ECZEMA (*Regional Forms, Hands*), SKIN, BACTERIOLOGY OR (*Cheirropompholyx* or *Dysidrosis*).

**Cheloid.** See CICATRICES (*Keloid or Cheloid*), SCLERODERMIA (*Circumscribed*).

**Chelonisomus.**—A teratological state in which there is eversion of the abdomen and thorax and excessive retroflexion of the body, turtle-like monstrosity (hence the name

chelonisomus, from *χελών*, a turtle, and *σῶμα*, a body), *schistosomus reflexus* (Gurli)

**Chelsea Pensioner.**—The name given to a laxative, containing sulphur, rhubarb, bitartrate of potash, guaiac, honey, and nutmeg; used specially in cases of hemorrhoids

**Cheltenham.** See BALNEOLOGY (*Great Britain, Chalybeate*), MINERAL WATERS (*Muriated Saline*)

**Chemical Trades.** See TRADES, DANGEROUS (*Chemical*)

**Chemosis.**—(Edematous swelling of the conjunctiva of the eye, which is thus raised and caused to project over the edge of the cornea. See CONJUNCTIVA, DISEASES OF (*Acute Ophthalmia*))

**Chemotaxis.**—An attraction or affinity of one tissue for a similar tissue, *eg* of nerve for nerve, also the power of attraction or repulsion which some cells (or some constituents of cells) show as regards other cells

**Chenopodium.**—The goosefoot, several species of which have been used for their medicinal qualities, the oil of the fruit of *C. ambrosioides* is official in the United States Pharmacopœia, and is used as a vermifuge, other species are used as diuretics, emmenagogues, narcotics, etc

**Cherry Laurel.** See DERMATITIS TRAUMATICA ET VENENATA (*Vegetable Agents*)

**Chest.** See AORTA, THORACIC, ANEURYSM (*Physical Signs*), ASTHMA (*Symptoms*), CHILDREN, CLINICAL EXAMINATION (*Physical Examination of Chest*), MEDIASINUM, MEDICINE, FORENSIC (*Infanticide*), SYRINGOMYELIA (*Symptoms, Thorax en bateau*), *vide infra* under CHEST, CLINICAL EXAMINATION, CHEST, DEFORMITIES, CHEST, INJURIES, CHEST-WALL, AFFECTIONS OF

**Chest, Clinical Investigation of the.**—In the clinical investigation of the chest the observer may gain much valuable information by a general survey of the patient, the appearance, expression, attitude, etc, before turning to the direct investigation of any cardiac or pulmonary derangement which may be suspected (Physiognomy). Yet the physiological functions and pathological alterations of cardiac and pulmonary organs are so intimately associated that it is impossible for an observer to differentiate how much of the patient's altered appearance and attitude may be due to disturbance of the functions of respiration and how much to disturbance of the circulatory organs, unless a careful physical examination of the chest be made, fully investigating the condition of the lungs and heart

In examining the chest it is necessary to have the patient as far as possible at rest, with the limbs and trunk symmetrical and the light falling equally upon the parts under examination

**INSPECTION.—FORM OF CHEST.—Normal.**—It is not common to meet the ideal typical chest, with a well-developed almost symmetrical bony frame, well clothed with muscle, fairly rounded on the anterior aspect, the interspaces visible below but not above, the obliquity of the ribs increasing from above downwards, and the epigastric angle nearly a right angle. Many alterations are met which cannot justly be considered pathological, thus as the result of occupation, *eg* the depression at the lower end of the sternum found in shoemakers. During health the respiratory and circulatory movements are accompanied by ceaseless changes in the outlines of the chest. With full inspiration, full expansion of the chest, the axes of the ellipse become approximately equal and the horizontal outline becomes more circular. The framework of the chest is shortened from above downwards, the ribs are raised and approach the horizontal plane, the intercostal spaces are narrow anteriorly, the epigastric angle is wide, the sternum is carried forwards and upwards, the shoulders are raised, and the scapulae are closely applied to the chest-wall. Such is the chest of full inspiration. With full expiration the transverse axis of the ellipse markedly exceeds the antero-posterior, the ribs are depressed, the framework of the chest is lengthened from above downwards, the intercostal spaces are wide anteriorly, the sternum passes downwards and backwards, the shoulders are low, and the scapular angles project somewhat from the chest-wall. Such is the chest of expiration. While these changes in the outline of the chest are constantly going on in health, in disease the same changes occur in an exaggerated degree. In disease and deformity there may be an increase or diminution in the length or height of the bony framework, an alteration in the horizontal position of the ribs leading to an alteration in the axes of the ellipse, an increase or diminution in the width of the intercostal spaces, in the size of the epigastric angle, in the position and direction of the sternum and spine, in the height of the shoulders and the position of the scapulae, and all these factors have a definite relation to the outline of the horizontal plane of the chest, and it is by the study of the outline of the horizontal plane that the observer will get the best idea of the alterations in the chest in disease and deformity. The outline of the horizontal plane of the chest is best ascertained by means of the cyrtometer. The best idea of the shape of the chest can be got by taking the outline at a given point with the cyrtometer. Various elaborate instruments are to be had, but a very good cyrtometer can

be made with two bars of malleable metal connected posteriorly by means of a rubber hinge. The two bars, being moulded round the chest, and their point of intersection marked, they can then be detached and placed upon paper and an accurate tracing obtained. The outline of the adult chest is seldom symmetrical. Employments which cause individuals to assume certain positions or to use a certain set of muscles constantly are very prone to cause variations in the shape of the chest, without these variations having any direct pathological significance. The circumference of the right side is, as a rule, larger than the circumference of the left, in a small number of cases the left is the larger. In the adult the lateral diameter exceeds the antero-posterior, in the child the chest is more circular in form, the two diameters being about equal.

*Alterations in Size and Shape, etc.*—In *emphysema* the chest presents a characteristic appearance, the neck appears shortened, the shoulders elevated, and the back rounded, there is a bilateral increase of the chest, the outline tending to assume the circular form seen in childhood. The chest is often described as "barrel-shaped." In the "alar" or "phthical" chest we find sloping shoulders, prominent thyroid cartilage, deep hollow over the episternal notch, and prominent scapulae. Here the antero-posterior diameter in the upper two-thirds is much shortened, and instead of a convexity there is usually flattening. The ribs tend to slope downwards instead of coming forward in a normal curve.

*Unilateral increase* of a general character is met with in effusion into the pleura. Localised increase may arise from acute croupous pneumonia, tumours of the lung, and mediastinal growth. Marked increase over the lower and right side of the chest should suggest the possibility of some hepatic affection, while on the left side the spleen should be remembered. In cardiac enlargement, especially when the enlargement has developed during childhood, there may be marked bulging or increase in the pre-cordial region, the bulging from aortic aneurism and pericardial effusion should also be borne in mind. The deformities of the chest (*q.v.*) may also cause bulging, especially the apparent unilateral alteration due to spinal curvature.

*Unilateral diminution* may result from chronic fibroid phthisis, or from old-standing pleurisy or empyema. If the fluid in the pleural cavity be not evacuated before changes have taken place in the lung and pleura, the corresponding side of the chest-wall becomes retracted, secondary to a collapse of the lung. *Local flattening* may be noticed below the clavicle as a result of diminished functional activity of the upper lobes, which is usually associated with some organic lesions of a tuberculous nature. In children depression of the chest-

wall may follow collapse or chronic interstitial pneumonia.

Before concluding the inspection of the chest the presence or absence of enlarged or pulsatory blood-vessels at the base of the neck should be observed. In *emphysema* and chronic bronchitis the vessels are distended, and a pulsation in the jugular veins is met with in cases of tricuspid regurgitation. Pressure on the superior vena cava and innominate veins from aneurism or intrathoracic new growth produces tortuosity of the veins above the clavicles and the engorgement of the veins of the trunk and limbs.

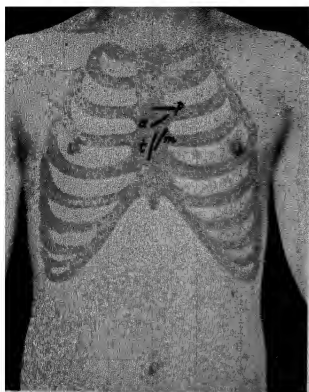
*The Movements*—1 *Lungs*—In health these are respiratory and circulatory. The points to note in regard to the respiratory movements are—*The rate of respiration.* The average of this is 14 to 18 per minute in the adult, in the child more rapid, about 40 per minute at birth, but gradually slowing as age advances. In health the ratio between heart-beats and respiratory movements is 4 to 1.

*Dyspnoea*, or difficulty in breathing, is a common and distressing condition in chest affections. Any cause which prevents the proper oxygenation of the blood in the lungs will produce the subjective feeling of difficulty of breathing, and the objective signs of increased respiratory effort. The error may be in the respiratory tract, sufficient oxygen not reaching the pulmonary capillaries, in an insufficient flow of blood through the lungs, as in valvular disease of the heart, in the blood itself being unable to take up sufficient oxygen for the needs of the body, as in advanced anaemia. *Dyspnoea* may thus be both *inspiratory* and *expiratory*. It may be for the most part expiratory, as in *emphysema*, when the lung has lost its elastic power, or when a tumour obstructs the glottis during expiration. *Inspiratory dyspnoea* is seen when there is obstruction to the entrance of air at the glottis, as in membranous laryngitis, spasm, paralysis of the dilator muscles of the glottis, etc. When from difficulty of breathing the patient assumes the upright position the condition is termed *orthopnoea*, the position being assumed to give the extraordinary muscles of respiration the fullest possible play.

2 *The rhythm of the respiratory movements* is in health perfectly regular; in disease this regularity may disappear. A peculiar type of irregularity, known as *Cheyne-Stokes' respiration*, may be met with in advanced cardiac and renal disease and in cerebral affections. In health the relative duration of inspiration and expiration is in the ratio of 5 to 6. In disease this proportion may be lost. If there be an obstruction to the entrance of air into the chest, inspiration will be prolonged. There is marked prolongation of expiration when there is difficulty in expelling the air from the chest, as in *asthma* and *emphysema*.

3. *The type of the respiratory movement* differs

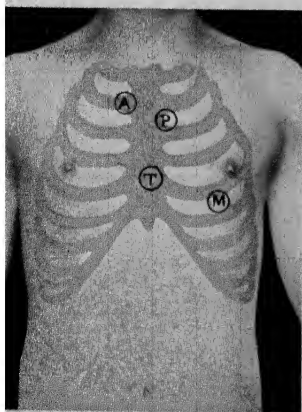




Position of the cardiac orifices in relation to the thoracic wall.

a, the aortic orifice,  
m, the mitral orifice.

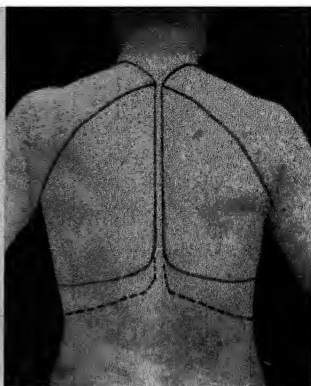
p, the pulmonary orifice,  
t, the tricuspid orifice.



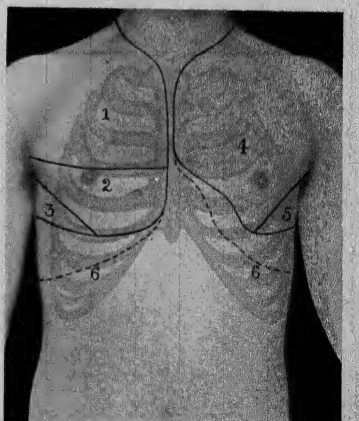
#### Auscultatory Areas.

A, the aortic area,  
M, the mitral area.

P, the pulmonary area,  
T, the tricuspid area.



Percussion outlines of the lungs posteriorly. The continuous black lines indicate the percussion outlines of the lungs and the oblique fissure on either side. The dotted lines show the line of reflection of the pleurae.



Percussion outlines of the lungs anteriorly. The continuous black lines indicate the percussion outlines of the lungs and the pulmonary fissures. The dotted lines 6 show the lines of reflection of the pleurae. The figures 1, 2, 3 indicate the upper, middle, and lower lobes of the right lung; 4 and 5 the upper and lower lobes of the left lung.



in the two sexes. In the male it is abdomino-thoracic, in the female thoraco-abdominal. In disease these types may be altered. When the action of the diaphragm and abdominal muscles is interfered with, as in abdominal tumours, ascites, pain in the abdomen, the movements may be purely thoracic, when there is marked pleural pain the intercostal muscles may be more or less fixed, and the breathing be abdominal. Pure abdominal breathing is seen in its fully developed form in paralysis of the intercostal muscles. Diminished local expansion is seen when one lung, or part of a lung, becomes functionless, as in pleurisy with effusion, pneumonia, phthisis.

4. The extent of the chest movement has finally to be observed. In the characteristic "alar" and "barrel-shaped chest," previously described, the movements are very slight. Deficient expansion or inspiration is an important diagnostic sign. When one side of the chest moves more markedly than the other we may find a pneumonia, pleurisy, or tuberculous consolidation in the motionless side.

*The Circulatory Movements.*—The apex beat, a gentle pulsation visible and palpable in the fifth interspace internal to the mammary line, extending a single interspace vertically, laterally about one inch, regular in rhythm, and systolic in time, is in health the only cardiac movement visible in the chest. In disease it may be displaced from its normal position, the whole heart being displaced, as in pleurisy with effusion, when the heart is pushed to one or other side, in ascites or abdominal tumour, when it is pushed upwards, in emphysema and mediastinal tumour, when it may be pushed downwards. By far the most common cause of dislocation of the apex beat is, however, to be found in the heart itself. Marked displacement downwards and outwards is seen in hypertrophy and dilatation of the left ventricle, as in aortic disease, in hypertrophy and dilatation of the right ventricle the displacement is more outwards, less downwards, in pericardial effusion there is an upward and slightly outward displacement. Besides the apex beat, other pulsations may be visible, thus in the third, fourth, and fifth interspaces to the left of the sternum when there is hypertrophy of the right ventricle, or when the chest-wall is thin and the lung retracted, as in fibroid phthisis, the pulsation of a normal right ventricle may be visible. Pulsation at the second and third right costal cartilages may be seen in aneurism of the aorta. In retraction of the lung the pulsation of the conus arteriosus of the pulmonary artery may be visible. The observer notes then the pulsation, its position, extent, character, and time in the cardiac cycle, and the presence or absence of abnormal pulsation.

*Lungs.*—*Palpation of the Chest-wall.*—The extent of the chest movements which have been

observed under inspection should be verified by palpation, requesting the patient to take a deep breath. The expansion of the two apices, the two infra-clavicular regions, the axillary regions, and the bases of the lung should be in turn carefully investigated. Areas of superficial tenderness may be detected during palpation, and should be carefully noted. The state of the intercostal spaces should be noted, and the presence of various accompaniments, such as friction fremitus, râles, etc., may also be determined. The vocal fremitus, or the ability of the thoracic viscera and chest-wall to transmit vibrations produced by the voice, must then be determined. This is subject to great variation consistent with health, being more marked in men than in women and children, and is diminished when the chest-wall is well covered with muscles and fat. Further, the vocal fremitus is greater on the right side than on the left, owing to the relative positions of the right and left main bronchi. The diseases causing a decrease of vocal fremitus are pleural effusion, great pleural thickening, pneumothorax, and any condition which causes occlusion of a large bronchus, such as a tumour or copious secretion of mucus. Increase of the vocal fremitus is met with in consolidation of the lung (pneumonia and tubercle) or over a cavity surrounded by consolidated lung or a tumour in the thorax intimately connected with a bronchus.

*HEART.*—Palpation of the chest-wall will also give information as to the position and character of the cardiac pulsation, and thus confirms what has already been observed as to the position of the apex beat and the presence or absence of abnormal pulsation.

The cardiac pulsation may be accompanied by a thrill—a gentle vibratile sensation which is felt on placing the hand over the region of the heart. A thrill is produced in the same way as a murmur from fluid waves being set up within the blood-stream, the resulting vibrations being sufficiently ample to pass through the chest-wall and be perceptible to the hand of the observer. If a thrill is present note its time in the cardiac cycle and its position and character. A thrill presystolic in time (the most common variety) is usually perceptible in mitral stenosis. Systolic thrills, as in mitral regurgitation or aortic stenosis, are fairly frequent, diastolic exceedingly rare.

In percussion of the lungs the observer begins above the clavicles and passes down the chest in the mammary line, laying the pleximeter finger along the interspace or rib, and not across. Compare corresponding points of the chest-wall on the two sides, for if this be not done, the first degree of dullness, comparative dullness, may easily be missed. In percussing in the mammary region percuss outside the mammary line; internal to the mammary line on the left side, the sound will be altered by the underlying

heart. When percussing posteriorly strong percussion must be used, except at the lower margins of the lungs. The patient should be bending slightly forward with the arms symmetrical and the head straight, for if the head be not straight, differing tension of the muscles over the apex will give rise to an alteration in the percussion sound.

The normal thoracic percussion sound got on striking over a thick layer of healthy resonant lung may be described as an ample percussion sound of considerable duration, low in pitch, and of a clear tone. In disease the resonance of the sound may be increased, the sound may be hyper-resonant. Slight hyper-resonance is got in emphysema, more marked on percussing over relaxed lung, as above a pleural effusion, or where exudation and air-containing tissue are intimately mixed, as in oedema, early pneumonia, etc. Marked hyper-resonance is met in pneumothorax, pneumopericardium. The resonance of the sound may be diminished—the sound is dull. Comparative dullness is got where there is some pleural thickening, in early phthisis, etc., dullness where the consolidation is more advanced, as in pneumonia, absolute dullness is found typically in effusion into the pleura. In some cases the percussion sound may have a special quality, as the crack-pot sound, the amphoric sound, the bell sound.

**Auscultation**—Next auscultate the lungs. Note the inspiratory and expiratory phase of the breath sound, their relative duration, their pitch and intensity, and the presence or absence of a pause between them, and thus deduce the type of the breath sound.

The normal type of breath sound heard all over the lung except at the interscapular region is vesicular. Inspiration is a gentle continuous rushing sound, expiration a thinner, fainter sound, which at times may not be audible, inspiration is three times as long as, and passes directly into, expiration. In disease this vesicular murmur becomes altered. The breath sound may be harsh, as heard in the normal child's chest, or over the healthy lung when the other is rendered functionless, as in pneumonia or pleurisy with effusion. *Harsh vesicular breathing* with prolonged expiration is heard in slight consolidation of the lung, as in tuberculosis, the audible part of expiration being prolonged. Wavy, jerky, or interrupted vesicular breathing may be nervous in origin, but it usually results from delay in the entrance of air into the pulmonary bronchi through catarrh giving rise to obstruction, and when localised is to be regarded with suspicion. Clinically it is found in early tuberculous infiltration, bronchitis. *Faintness or absence of the breath sounds* may be present if the sound vibrations produced at the larynx and upper part of the respiratory tract are cut off by obstruction of a bronchus, effusion into the pleural sac, thickening of the pleura, etc.

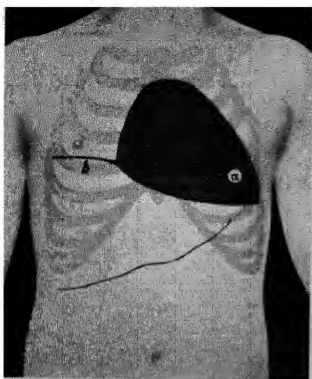
When in disease the sound vibrations produced at the naso-pharynx, pharynx, and larynx are not damped down by passing through spongy air-containing lung, the sound over the chest has the same characters as at its point of production, and, being similar to the sound heard on listening over a bronchus, is called *bronchial*. Inspiration and expiration are about equal in length, they are separated by a distinct pause and have a blowing quality. Three kinds of bronchial breath sounds are recognised, distinguished according to their pitch. High-pitched or tubular breathing, medium-pitched or simple bronchial, low-pitched or cavernous. The simple or medium-pitched variety is heard where the lung is a better conductor of sound, as in consolidation in phthisis. The high-pitched variety, more intense and harsh in character, is found where consolidation is more complete, as in croupous pneumonia. The low-pitched or cavernous variety differs from the other varieties in its pitch and in having a distinctly hollow quality. It is heard over cavities in the lung, phthisical or bronchiectatic.

**Broncho-vesicular Breathing**—The type of breath sound heard in health in the interscapular region and over the manubrium sterni possesses some of the characters of bronchial and some of the characters of vesicular breathing. Pathologically it indicates some slight increase in the conducting power of the lung, as in early tuberculous infiltration.

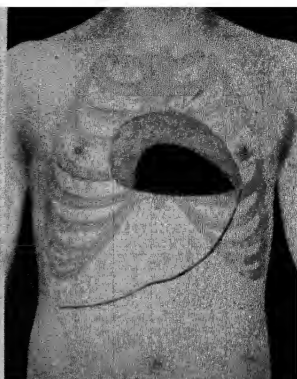
**Amphoric breathing**, a rare variety, has a hollow whistling character. Like bronchial, it may be high, medium, low pitched. It may be heard over a pneumothorax where there is free communication between the pleura and a bronchus, or over smooth-walled cavities having free communication with a bronchus.

**Accompaniments**—Having determined the type of the breath sounds, notice the presence or absence of accompaniments. If present they may be extra-pulmonary, such as friction. Intra-pulmonary accompaniments may be dry râles or rhonchi, snoring, cooing, whistling, or creaking sounds due to an alteration in the lumen of a bronchial tube, as in asthma or bronchitis, or there may be moist râles or crepitations, sounds which convey to the ear the idea of the bursting of small bubbles or the crackling of hair rubbed between the fingers.

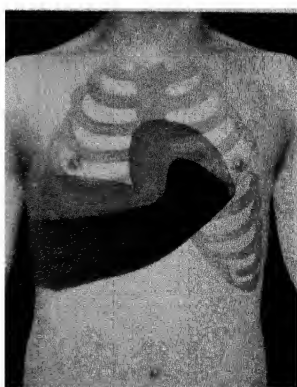
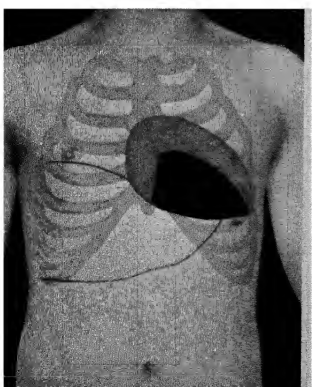
Examine next the *vocal resonance*, asking the patient to repeat ninety-nine, or one, one, one. In health the sound vibrations are conducted to the stethoscope altered by their passage down the column of air in the respiratory tract and through the chest-wall. In disease when the lung is consolidated, as in phthisis, pneumonia, the sound is increased. When the sound is markedly increased, it is called *bronchophony*. The sound vibrations are diminished or lost when there are diminished facilities for their conduction, as when a bronchus is plugged,



of cardiac dullness in case of pericarditis with effusion. *a*, the apex-beat; *b*, the upper margin of the liver.



Superficial and deep cardiac dullness in enlargement of the right auricle.





when there is thickening of the pleura, effusion into the pleura, cedema of the chest-wall, etc.

Under certain conditions the whispered voice is well heard, as if spoken into the end of the stethoscope. This is known as *whispering pectoriloquy* (see article "Lungs (Phthisis)")

**PERCUSSION OF HEART**.—Turning next to the *percussion of the heart*, two areas of dulness are recognised the superficial or area of absolute dulness, the deep or area of relative dulness. The *superficial cardiac dulness* corresponds to that layer of the heart which lies uncovered by lung, having a truncated pyramidal shape, with its right border along the left border of the sternum, its upper boundary at the fourth costal cartilage, its left border about the parasternal line. The size of the area varies with the state of expansion of the lung. In enlargement of the heart it is increased, in emphysema it is diminished or absent, the over-expanded lung occupying the area. The area of *deep cardiac dulness* is of much greater importance to the clinician. Using strong percussion, percussing from without inwards along the third and fourth right interspaces, the right border of the heart is found about two inches to the right of the mid-sternal line. The left border is next mapped out, percussing along the interspaces from without inwards, beginning outside the mammary line, where a clear thoracic percussion sound is obtainable. By marking in each interspace when the relative dulness is first reached and joining the points, the position of the left border of the heart can be obtained. In health it is usually in the mammary line, that is about  $3\frac{1}{2}$  inches to the left of the mid-sternal line at the level of the fourth interspace. In percussing the deep cardiac dulness, much assistance will be got by taking into consideration the sense of resistance when carrying out the percussion. The cardiac dulness is found to be increased in cases of dilatation and hypertrophy and in dilatation. In hypertrophy and dilatation of the left side the increase in the area is downwards and outwards, in hypertrophy and dilatation of the right side the increased dulness is more in the transverse direction. In effusion into the pericardium the dulness takes the shape of the pericardial sac, it is somewhat pear-shaped, the stalk of the pear being upwards. It is sometimes, as in emphysema, impossible to map out the cardiac dulness with certainty. A good deal, however, will depend upon the skill of the observer. Accurate percussion of the heart requires much practice.

**Auscultation**.—Taking next the auscultation of the heart, the different cardiac areas are listened at in turn, beginning, as a rule, with the mitral, and the character of the sounds is noted. Are the sounds healthy? If not healthy, is the alteration quantitative? Are the sounds louder or fainter than in health? or is the alteration

qualitative? Is there a murmur present? Quantitative alterations may be various, the first sounds may be short, sharp, and accentuated, as in dilatation, it may be doubled, a somewhat rare condition, it may be prolonged and of grave tone, as in the hypertrophy and dilatation of chronic kidney disease. The second sound may be loud and accentuated at either aorta, it may be faint, or it may be doubled.

Is the alteration in the sounds qualitative? Is a murmur present? If so, time the murmur in the cardiac cycle—systolic, diastolic, or presystolic (auriculo-systolic), ascertain its point of differential maximum intensity, its sound character, duration, and the direction of propagation.

**Extra Cardiac Areas**.—Having auscultated the heart, the extra cardiac areas are noticed, the upper part of the sternum, the roots of the neck, and the epigastric region.

**Palpate** the upper part of the sternum for any pulsation or thrill, and percuss to ascertain if there is any increased impairment of the percussion sound, as in aneurism or dilatation of the aorta. Notice the root of the neck for any swelling, undue pulsation, and for venous pulsation, as in tricuspid regurgitation. The epigastric region may show pulsation, tumour, etc., which may have important bearing upon the condition of the heart.

In many cases the simple clinical examination as sketched is insufficient.

Extra auscultation will frequently give valuable information. Note the cough, its character, frequency, etc.

Note the presence or absence of expectoration, its amount, reaction, appearance, naked-eye and microscopic, etc.

In cases where effusion into the pleura is suspected, or where the nature of an existing effusion is in doubt, it is often advisable to explore with the needle of an exploring syringe or aspirator.

In cardiac cases useful information may be got from the sphygmograph, cardiograph, sphygmomanometer, or sphygmometer.

The Röntgen rays have been applied to the diagnosis of respiratory and cardiac disease, but so far there are a number of difficulties in their application.

Controlling evidence is of much importance in many diseases of the chest. Examination of the larynx is of the greatest importance in cases of early phthisis, suspected aneurism.

The condition of the blood, the red corpuscles, the leucocytes, and the hemoglobin are all of importance in cardiac and respiratory disease. Thus the presence of anæmia may account for a cardiac murmur, which otherwise must have been considered organic, the presence of a leucocytosis may give confirmatory evidence of a deep-seated patch of pneumonia.

## Chest, Deformities of.

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In describing deformities of the chest it is most convenient to consider them in relation to their causal factor. The deformity may be the result of a developmental error, or it may be acquired. If acquired, it is the result of disease of the parietes, of the thoracic organs, or from the effects of external mechanical causes.

### (i.) CONGENITAL DEFORMITIES OF THE CHEST.

—Congenital deformities of the chest are comparatively rare. Cleft sternum results from deficient union of the visceral layers in the embryo. If the fissure be marked, ectopia cordis results. Defects of the lateral or posterior thoracic wall generally arise as the result of amniotic adhesions, and protrusion of the lung may then take place.

(ii.) DEFORMITIES DUE TO ALTERATIONS IN THE PARIETES.—The deformities of the *spinal column* produce a deformity of the thorax, slow, progressive, and at times considerable. In *scoliosis* or *lateral curvature* of the spine, the curvature of the vertebral column modifies little by little the direction of the ribs. The curvature of the ribs is increased on the side of the vertebral convexity, diminished on the side of the vertebral concavity, and, as a consequence, there is a protrusion of the chest on the side of the spinal convexity and a depression on the side of the concavity, and on the side of the spinal concavity the ribs approach each other, and may even overlap. The horizontal outline of the chest assumes an ellipsoidal form with a posterior protrusion of the ribs on the side of the scoliotic convexity, and a projection of the anterior angle of the ribs on the opposite side. The half of the thorax corresponding to the convexity is diminished in capacity; the opposite half retains its sectional capacity, but is diminished in vertical measurement. The shoulder on the side of the scoliotic convexity is markedly elevated, the scapula is thrown out behind, its angle is elevated, and is carried out from the middle line. In a number of cases a very complicated deformity is produced as the result of curves of compensation in the vertebral column, but there is always a diminution in the capacity of the thorax on the side of the scoliotic convexity, and as a result of this the action of the heart is impeded from displacement, and increased work is thrown on the right side in carrying on the pulmonary circulation.

*Kyphosis* or *backward arching* of the spine produces a flattening of the sides of the chest, and an increase in the antero-posterior diameter

of the chest. The sternum becomes curved about its middle, and there thus results an anterior convexity or arching, more rarely an anterior concavity of the chest. The infra-clavicular depressions are exaggerated, the inferior angles of the scapulae project from the chest-wall in the "alar" fashion, the abdomen frequently assumes an anomalous anterior projection.

In *Pott's disease* or *angular curvature* of the spine the deformity varies with the position of the spinal curve. If the curve be in the upper dorsal region the thorax is flattened from before backwards, the antero-posterior diameter of the horizontal section is diminished. When the projection is in the inferior dorsal region the thorax is flattened transversely, the lateral diameter of the horizontal section is diminished, and the outline of the section assumes a circular form.

In *osteomalacia* the deformities of the chest are secondary to the incurvation of the spine and the softening of the ribs, and in advanced cases may be very complicated.

In *osteitis deformans* the spine, more or less ankylosed, is inclined forwards in a dorsal lordosis. The ribs are fixed posteriorly, the chest is flattened laterally. The respiration is impeded, and is almost purely diaphragmatic.

In *acromegaly* the chest is projected forwards, the antero-posterior diameter of the horizontal plane is increased, the lateral aspects flattened, and the lateral diameter diminished. The anterior projection is most marked in the lower sternal region, and thus the sternum lies obliquely with its anterior surface directed forwards and upwards. The sternum is thickened, widened, and elongated. The anterior surface shows a series of grooves. The angulus Ludovici is very prominent, the xiphisternum is elongated and ossified. The clavicles are enlarged and the extremities thickened. The ribs are massive, so increased in bulk that their edges may be almost in contact, obliterating the intercostal spaces; the costal cartilages, broadened and ossified, form a chaplet, which may pass beyond the plane of the anterior aspect of the sternum. The spine may show a pronounced dorsal kyphosis. The whole bony framework of the chest is hypertrophied, and the respiration is impeded, being abdominal in type.

In *rickets* the costal cartilages are abnormally firm, while the ribs are softer than natural, especially at their enlarged growing ends. The deformity is characterised by the formation of a groove running down the chest-wall almost parallel to the direction of the sternum, situated outside the junction of the rib with its cartilage, extending down to the costal margin. There is an increase in the antero-posterior diameter of the sectional outline, the sternum being carried forwards, and a diminution in the transverse diameter. In addition to this alteration in the



outline of the chest, there is an enlargement of the cartilaginous ends of the ribs, which results in the beaded appearance known as the "rickety rosary." The deformity is the result of the atmospheric pressure acting upon the softened ribs. When the diaphragm descends during inspiration the rickety softened ribs are unable to withstand the atmospheric pressure during the time the fresh supply of air is entering the lung. The chest-wall yields at its softest part, that is, immediately external to the enlarged growing ends of the ribs, and a sulcus forms down the sides of the chest, while the sternum is carried forwards by the yielding costal cartilages. The rickety deformity may form without any actual impediment to the entrance of air into the chest, but is accentuated if any impediment such as a bronchial catarrh be superadded to the rickets. The deformity being due to an undue softness of the chest-wall, the solid organs subjacent may modify the deformity; thus the liver supports the lower chest-wall on the right side, while the heart may form a prominence on the left side.

In *xyriomyelia* a boat-shaped hollow sometimes develops in the upper part of the thorax, the region below the level of the fifth rib being normal. The head is buried between the shoulders, and the sternum shows considerable obliquity and the shoulders are carried forwards. There is no functional disturbance.

(iii) DEFORMITIES OF THE CHEST DUE TO MECHANICAL CAUSES.—Any more or less continuous alteration in position or increased pressure will affect the outline of the thorax and give rise to greater or less deformity. Elevation of one shoulder, usually the left, is met with in clerks who, on account of the position taken while writing, tend to have a slight lateral spinal curvature with elevation of the shoulder. Again, in persons whose occupation necessitates the carrying of heavy weights on one arm, the opposite shoulder takes up a higher position, and a slight deformity is induced. In some trades considerable deformity may result from pressure, as, for example, the well-recognised depression which occurs at the lower end of the sternum in shoemakers from the pressure of the "last." In the developing chest of young boys a projection of one or more costal cartilages may result from lateral compression in athletic exercises. The cartilage, being soft when it is compressed between the rib and the sternum, bends, and an angular projection forms. In the female, mechanical modification may result from tight corsets. The lower ribs are forced inwards and imprint their traces on the viscera, the massed intestine presses upon the pelvic organs, the descent of the diaphragm is limited, and the respiration becomes thoracic in type, the respiratory capacity of the lower part of the chest is diminished, and the epigastric angle is reduced. The pressure results in entoptysis,

or else, if dislocation of the different organs be not produced, an indelible impression remains with an alteration in the shape of the organ.

(iv) DEFORMITIES DUE TO ABNORMAL CONDITIONS OF THE THORACIC ORGANS.—The framework of the chest undergoing, as it does, ceaseless temporary changes in outline during the respiratory and circulatory movements, the shape of the outline must largely depend upon the condition of the contained viscera. Any alteration taking place in the thoracic viscera must to a greater or less extent tell upon the parietes. The development of the chest must be dependent upon the development of the contained organs, and any want of respiratory capacity will tell in the most marked manner upon the form and outline of the chest. While thus departing from the normal form may not amount to absolute deformity, but the abnormality is of importance to the physician as showing the existence of past or the probability of future lung mischief. Of such a type is the "alar" or "pterygoid" chest. In such individuals, as the result of improper surroundings and education during early life, there is deficient lung development, or it may be that there is a congenitally small lung capacity. As a consequence there is deficient development of the chest, it is shallow and narrow, there is an increased obliquity of the ribs which results in a proportional diminution of both the antero-posterior and transverse diameters, the increased obliquity of the ribs results in a drooping and forward inclination of the shoulders, the upper part of the scapula is thus carried forwards and the angle tilted backwards from the ribs, giving the chest the peculiar "alar" appearance. While the increased obliquity of the ribs results in increase in the vertical length of the bony framework, the vertical capacity of the chest is really diminished by an increase in the height of the diaphragm.

The *flat chest*, the type of the tuberculous, shows a marked and distinctive deformity. There is a loss in the normal anterior rounding of the chest, there is a diminution in the antero-posterior diameter of the sectional outline in the marked form, the cartilages of the true ribs having a diminished anterior curve, in some cases the sternum even being depressed. In the flat chest, as distinguished from the alar, there may be no increased obliquity of the ribs. Both alar and flat chests are essentially deformities the result of deficient lung capacity, which is said by some to be congenital, but may also be the result of deficient pulmonary development arising from adverse circumstances and surroundings.

The *pygma* chest is characterised by an alteration of the almost circular sectional outline of the childish chest to a triangular form. There is an increase in the antero-posterior diameter of the chest due essentially to a straightening of the true ribs in front of their angles, and the

sternum is thus carried forwards. The *transversely constricted* chest is a very common form of deformity. The chest shows a depression or groove which, commencing at the level of the xiphisternum, passes outwards and downwards till it shades away towards the mid-axillary line. The transversely constricted and pigeon deformities have essentially the same causal factor—an impediment to the entrance of air into the lung during the developmental period of life, while the chest-wall is soft and yielding. Given an affection of the respiratory tract which will interfere with the free entrance of air, such as enlarged tonsils, bronchitis, whooping-cough, etc., and either deformity may result. The respiratory obstruction causes inspiratory dyspnoea with increased efforts at inspiration. The chest and lung are more easily expanded above than below, hence when the diaphragm descends and the ribs at the upper part of the chest are drawn up, the lungs not expanding fully at their lower part, there is a tendency to form a vacuum at the lower part of the chest, and the atmospheric pressure forces in the chest-wall and a sulcus is thus formed. The sulcus corresponds to the upper level of the abdominal organs which support the lower part of the chest-wall. The transversely constricted chest is thus developed. When the inspiratory dyspnoea has been more pronounced and more persistent, the deformity advances to the pigeon chest. Forced inspiration over-expands the upper thorax, protrusion of the sternum takes place, the atmospheric pressure forces in the lower part of the chest, producing the transverse sulcus and a bending back of the xiphisternum. From the altered position of the sternum the costal cartilages become straightened in front of their angles, and the greatest transverse diameter of the chest lies posterior to the normal position.

The *emphysematous chest* is characterised by an increase in all the diameters of the sectional outline of the chest—from the ellipse the outline assumes a circular form. Repeated and prolonged inspiratory efforts produce an over-distension of the lung, the lung becomes emphysematous. The lungs have become too voluminous to be accommodated within the fixed periphery of the chest when in an ellipsoidal form, the fixed periphery assume a circular outline to give a greater contained area for the enlarged lungs. The outline of the chest is circular, there is an increase in all the diameters, the sternum is arched, the shoulders are raised, and the spine is arched, the chest is fairly described by the epithet "barrel-shaped." The enlargement of the chest may be confined to the parts above the level of the xiphisternum, but frequently the whole chest is affected, in which case the epigastric angle is markedly enlarged.

*Unilateral alteration in the shape of the chest* may be seen in a variety of conditions affecting the lung or pleura.

A unilateral dilatation of the chest is rarely seen in the healthy side when disease impedes the action of the other lung. On the diseased it is markedly apparent by measurement or inspection in pleurisy with effusion, pneumothorax, and in cases of tumour of the lung. The intercostal spaces are obliterated, at times they may even bulge. Movement on the affected side is diminished, exaggerated on the sound side.

Diminution of one side of the chest may form a very striking deformity. It may follow upon chronic fibroid and destructive changes taking place in one lung, or upon pleurisy with effusion or empyema. In pleurisy or empyema, while the fluid is present, the lung is collapsed, if before the fluid be evacuated changes take place in the lung and pleura, preventing the expansion of the lung when the fluid ultimately becomes evacuated or absorbed, marked deformity results. The lung can no longer expand to fill up the affected side, the heart passes over towards the affected side, the opposite lung becomes emphysematous, and the atmospheric pressure forces in the chest-wall on the affected side. In such a condition there is marked deformity and diminution on the affected side, and the heart is displaced towards it. Marked deformity of a similar nature may result in chronic fibroid phthisis.

Local deformity of the chest-wall is frequently met. Local bulging or increase may be seen in encumbered pleural effusions, in tumour of the lung, in haema of the lung, in cardiac hypertrophy, in aneurysmal tumour.

Local contractions or diminution of the chest-wall due to intrathoracic disease is seen in phthisis even without the formation of a cavity, though it is more marked if a vomica be present.

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#### SURGICAL AFFECTIONS OF THE CHEST-WALL.

1 **SOFT PARTS**—CONCUSSION AND CONTUSION of the chest without evidence of injury to internal organs are of frequent occurrence, and in many cases lead to no serious results, but it must be borne in mind that an injury which appeared slight at first may later give rise to alarming symptoms. In concussion from the buffers of a railway carriage or the pressure of part of an ordinary carriage on the chest-wall very little may be observed at the time, and later extensive hemorrhage from an injured internal organ may lead to serious complications and death. In adults this is often observed than in children, as their chests, being more yielding, bear concussions much better. The shock from slight concussions is often much greater than might be expected, because the influence produced by them varies greatly both in adults and children, and cases are on record where a slight blow on

the chest has caused profound shock and sometimes death. It is therefore extremely important to treat all cases of concussion and contusion of the chest, even when slight, with the greatest care, avoiding prolonged examination during the period of shock. Laying the patient flat in bed with warmth judiciously applied round him, using stimulants in small quantity and often repeated, with subcutaneous injections of strychnine and ether where the tendency to heart failure is great, will generally be sufficient in ordinary cases of shock. As very serious injuries internally may result from blows on the chest without any external wound or any fracture of the ribs, it is important to keep the patient at rest for eight or ten days, by which time it may be assumed that the dangerous period is past.

Ordinary wounds not involving penetration into the chest, such as cuts and stabs only passing through skin and muscle, occur frequently, and should always be treated with the same antiseptic precautions as wounds elsewhere. When of small size, after thorough cleansing, a simple collodion dressing may be sufficient, but where extensive, sutures are required and aseptic or antiseptic dressings, according to the opinion of the surgeon in charge. In lacerated wounds and in very slight wounds with a large amount of extravasated blood in the tissues special care ought to be taken in the cleansing process, as should suppuration take place it spreads with great rapidity in the cellular tissue of the chest. In all such cases, and where wounds involve the muscles, it is important to note that the fixing of the dressings is so arranged that the arm is bandaged to the side, thus securing rest to the injured muscles. At the end of ten days the parts are generally sufficiently healed to allow the arm free of the bandages, and passive motion is employed to prevent stiffness.

*Wounds involving both the thorax and abdomen* are comparatively common, the vault of the diaphragm reaches the level of the fifth, and it may be the fourth rib on the left side, the pleura lines practically the entire wall of the thorax, hence a penetrating instrument is liable to traverse the pleural cavity and enter the cavity of the peritoneum. The injury to the abdominal viscera may be the more serious element, the organs most likely to be injured are the stomach, liver, spleen, and kidney. Perforation of the diaphragm on the left side may result in one or other of the forms of diaphragmatic hernia (see "Diaphragm, Surgical Affections of").

**2 THE RIBS AND STERNUM** — *Contusions* of the ribs are very common, and are caused by direct violence, producing frequently very persistent pain over the site of injury. If this pain does not yield to soothing fomentations with belladonna or laudanum for forty-eight hours, and thereafter the application of strips

of ordinary sticking plaster applied for a week, a small fly blister over the site of pain is very frequently of great service.

*Fractures* occur generally from the fourth to the eighth ribs, the upper and lower ribs being more protected, the floating ribs yield more to injuries, and consequently are less often broken. The most frequent seat of fracture is usually at one or other end of the rib, and the ribs most frequently broken are the fifth and sixth. A rib may be broken in two places, or may simply be cracked. The diagnosis is not always easy, a simple fracture being most easily felt by placing the hand flat on the chest and directing the patient to take a long breath. Sometimes placing two fingers over the site of pain and pressing one inward enables crepitation to be made out. It is a good plan to examine carefully each rib with the fingers, in order, if possible, to make out crepitation. Sometimes this is so difficult that it is not possible to be absolutely certain as to whether the rib is broken or not, but if the patient has intense pain at the end of inspiration or in turning suddenly in bed, and if his respiration is short and hurried, we are justified in concluding that he has had one or more ribs broken. Ribs are generally broken by direct violence, but a number of cases are on record where after violent sneezing or coughing one or more ribs have given way, and in the insane simply turning in bed has been known to fracture several ribs due to disease of the bone. Fractures of the ribs cause serious complications, with which we will deal later on when speaking of injuries to the viscera. The treatment of a simple fracture consists in strapping the injured side and applying a broad bandage round the chest, and where this gives comfort it may be continued, in certain cases, however, patients are easier without any bandage at all. The fracture is generally united in three weeks, and it is unnecessary to keep patients in bed in uncomplicated cases for longer than a few days. The *costal cartilages* are sometimes broken by direct violence, or a severe crush of the chest may lead to several costal cartilages giving way, and the same treatment is employed as for fracture of the ribs.

*Dislocations* of the ribs — Separation of the head of a rib from its articulation with the spinal column is exceedingly rare, while dislocation of a costal cartilage from the sternum is very uncommon. Cases have been mentioned both of forward and backward displacement, the forward being easily reduced by pressure, the backward requiring pressure on the sternum while the patient takes a deep breath.

*Sternum* — Fractures of the sternum may be transverse or oblique, and are caused either by direct or indirect violence. They occur between the manubrium and the gladiolus, or at the ensiform cartilage. The manubrium is generally

displaced backwards and behind the gladiolus, and is reduced when possible by placing the patient on the back, putting the knee between the shoulders, and pressing the ribs and gladiolus downwards. Sometimes it is impossible to reduce the displacement, and unless the manubrium is giving rise to uncomfortable pressure symptoms, operative interference is unnecessary. The ensiform cartilage may be driven backwards towards the spine by direct violence, and from pressure on the stomach vomiting may become so persistent that an operation to replace the displaced cartilage becomes a necessity. It is well to bear in mind that in both fractured ribs and sternum the intercostal and internal mammary arteries have been injured and the patients have died from fatal hemorrhage. Compound fractures of the ribs and sternum are dealt with in exactly the same way as compound fractures elsewhere. It is sometimes necessary to remove portions of rib in such circumstances, and the pleural cavity is as a rule penetrated, and therefore care must be taken to ensure careful drainage.

3. THE VISCERA.—Wounds of the lung lead to various symptoms, according to their extent, and may be produced by simple concussion, fractured ribs, stabs by knives, swords, and such like weapons, or by gunshot wounds or any projectiles having sufficient force to penetrate the chest-wall. Hemoptysis, emphysema, pneumothorax, hemothorax, are the symptoms which occur soon after an accident, while some days after an injury to the viscera we may have pneumonia, abscess of the lung, bronchitis, gangrene, or hernia of the lung.

*Hemoptysis* to a slight extent may occur in concussion or slight injury to the substance of the lung by a broken rib, and is frequently accompanied by *emphysema*, which consists in the air passing into the cellular tissue through the opening in the lung, and is diagnosed by placing the hand over the chest and feeling the crackling caused by the air in the cellular tissue. Where the wound in the lung is extensive, as by a sword thrust, the emphysema may extend over the whole body, and the patient may become unrecognizable.

This condition, however, is not very common. The treatment of moderate emphysema consists in strapping the chest in cases of fracture and dressing the wound antiseptically where it has occurred from a stab. Where, however, the emphysema is general, free incisions must be made into the cellular tissue to allow the air to escape, not forgetting the importance of the incisions on each side of the larynx, as the pressure in that region is so great as to threaten suffocation, and patients have died from neglect of this precaution.

*Pneumothorax*.—In some cases of injury to the lung, instead of emphysema occurring, the air rushes into the pleural cavity, compressing

the lung and giving rise to the most distressing dyspnoea, which is relieved by passing a trocar and canula into the pleural cavity and allowing the air to escape, having previously of course used antiseptic treatment for the skin. After the air has escaped a rubber tube with a flange (to prevent its passage into the chest) is introduced and antiseptic dressings applied.

*Hemothorax*.—In many cases in addition to air in the chest a large quantity of blood may exist from injury to the vessels in the lung. Where blood alone is extravasated the condition is called hemothorax, where both air and blood are present it is called hemo-pneumothorax. These cases are generally so serious that no interference is possible, and patients generally die from the extensive hemorrhage. Venesection is recommended in cases of hemorrhage in order to produce faintness and possible arrest of the bleeding. A certain number of cases recover from arrest of hemorrhage and absorption of the fluid. The balance of surgical experience points to the importance of not interfering unless there is evidence of empyema. Where there is a wound into the chest with free hemorrhage the question of opening up the wound, turning out the clots, looking for the bleeding vessel or vessels, and if possible securing them, must be considered and carried out in certain cases. Sometimes the hemorrhage is so great on turning out the clots from the chest that it is better to stuff the cavity with gauze.

*Traumatic hernia of the lung* consists in the protrusion and often the strangulation of a portion of the lung through a wound in the chest-wall. It is usually met with in wounds which open into the pleural cavity without involving the lung itself. The wound in the chest-wall must be of a certain size to allow of the protrusion of the lung, and it must correspond in position with one of the borders of the lung or with the corner of one of its lobes. The hernia may take place as soon as the penetrating weapon is withdrawn, or it may not take place for many hours after the injury. Violent expiratory movements have a good deal to do with its production, part of the air expelled from the lung on the healthy side may be driven into the partially collapsed lung on the injured side, so that it becomes distended and may protrude at the wound. If, on the other hand, the lung itself is wounded, and especially if one of the bronchi has been opened into, the air simply escapes into the pleural cavity. In the first instance, it is easy to return the protruded portion of lung, but after a time it tends to become strangulated, congested, oedematous, and irreducible, and may finally become gangrenous and slough away. Healing is then quite satisfactory as a rule, the wound in the chest-wall is closed with scar tissue, and the lung remains firmly adherent to the parietes.

*Bullet wounds of the lung*, as met with in civil practice, are usually caused by shots from a revolver. They have little penetrating power, and rarely traverse the thorax as a whole, they may lodge in the lung, or in the posterior wall of the chest, often beneath the skin. The injury produced resembles that caused by any other penetrating instrument. The wound in the skin is small, and gives exit to a little ooze of blood. The features resulting from injury to the lung depend on its situation and extent, the outlying portions may be wounded without any definite symptoms, penetration of the root of the lung or its vicinity may result in pneumothorax, emphysema, hæmoptysis, hæmorrhage, etc. The presence of the bullet, as a foreign body in the chest, does not appear to add to the gravity of the injury. The rule is not to be too eager to follow the course of a bullet in the chest, unless it is easily felt or is producing symptoms of irritation.

The large calibre and slow velocity of the rifle bullet of former days were attended with more extensive injury of the parietes, and were more likely to be complicated by the carrying in of portions of clothing, splinters of bone, etc., and the wound of the lung was often extensive, lacerated, and infected. With the modern rifle and bullet there is more likely to be a clean small hole right through the chest.

*Wounds of the heart and pericardium* are usually produced by pointed instruments or by bullets which penetrate the wall of the chest, there are certain rare cases on record in which a pointed foreign body in the œsophagus has penetrated the heart-wall.

The wound of the chest-wall is usually in the precordial region, and may appear to be of very little importance. The pericardium may alone be wounded, probably when it is on the stretch between its two attachments. The wall of the heart may be penetrated or perforated, the point of the weapon may be arrested in the myocardium of the ventricles, or may enter one or other of the cavities of the heart. The nature of the wound varies with the instrument causing it, the puncture of a needle is readily filled with clot, a stab with a knife may give rise to a wound which gapes, both ventricles may be penetrated, the apex of the heart may be cut off, a bullet may pass right through two or more cavities, or it may lodge in one of the latter, a bullet of high velocity may so raise the hydraulic pressure in the cavities of the heart that their walls may be extensively ruptured.

The pleura and lung are very commonly injured at the same time as the heart, especially the anterior reflection of the pleura on the left side. The great vessels of the chest may also be involved, *e.g.* aorta, vena cava, pulmonary vessels, azygos vein, etc. A wound of the heart may interfere with its functions in various ways,

its action may be arrested altogether by the injury to its walls or to its valves, or by the influence of the injury on its nerve mechanism, so that the individual dies of syncope, accumulation of blood in the pericardium (hæmopericardium) is a very constant accompaniment of wounds of the heart, it may be derived from one of the coronary arteries or from one of the cavities, if it does not escape externally from the wound in the pericardium, it accumulates and presses directly on the heart, so that the latter may cease to beat, if it does escape through the wound in the pericardium, the patient may bleed to death.

If the pleura has also been wounded, the blood may pass into the pleural cavity and result in an extreme form of hæmorrhage.

Should the patient survive, he may become the victim of septic complications, of which purulent pericarditis and pleurisy are the most important examples.

The symptoms associated with the lesions described are very variable and inconstant. The patient may present no evidences of serious injury, he may be able to stand, or even walk, more often there is a condition of syncope or collapse, which may be rapidly fatal, there may be external hæmorrhage sufficient to cause death in a few minutes, or there may be very little or none at all. The pulse is small, rapid, irregular, and intermittent, the heart sounds may be faint or inaudible, or they may be replaced by murmurs of very varied character, an increase in the area of the cardiac dulness may indicate hæmopericardium. Dyspnoea is very common, and may culminate in asphyxia, it may depend on the insufficiency of the pulmonary circulation resulting from the feeble and injured condition of the heart, or it may be due to complications such as hæmorrhage and pneumothorax.

Delirium, convulsions, paralysis are occasionally observed, the latter one usually the result of cerebral embolism.

The treatment of wounds of the heart does not differ from wounds of the chest in general. Absolute rest is essential. It may be advisable to bleed from the arm. If there be a portion of needle or other pointed instrument projecting from the wound in the chest, it should be extracted slowly by alternate movements of traction and rotation, so as to favour the clotting of blood in the track made by the instrument. Should there be an accumulation of blood in the pericardium it may be advisable to evacuate it, the use of a trocar and cannula for this purpose is condemned because of its incapacity to evacuate blood which has clotted, it does not allow one to identify and secure the bleeding-point, and its use entails the risk of wounding the heart or the pleura. Incision of the pericardium by open operation is the procedure recommended, great care must be taken not to open into one or other of the pleural cavities,

especially the left; the soft parts are reflected in the form of a flap having its base at the right border of the sternum, the cartilages of the third, fourth, and fifth left ribs are removed with or without a portion of the sternum. The pericardium is then exposed, and may be opened and evacuated, any bleeding-point is seized and ligatured, a wound in the heart-wall may be closed with interrupted sutures, silk is usually preferred because it is more durable than catgut, the sutures should not include the endocardium. Having arrested the hæmorrhage, the wound in the pericardium is sutured, leaving a small opening for drainage, occupied by a strand of gauze. Any pleural complication is thus dealt with before closing the external wound. Should the patient recover, he should be prohibited from attempting any exertion for several months, for fear of the scar yielding and causing aneurysm or rupture of the heart-wall.

Needles have been found in the heart after death, having given rise practically to no symptoms, on other occasions alarming symptoms have arisen from the passage of a needle through the heart. In one case in which a needle was removed a distinct bruit was audible previous to the operation, and disappeared immediately after.

### Chest-Wall, Affections of.

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1 ACUTE CELLULITIS AND ABSCESS OF THE CHEST-WALL.—This may involve the subcutaneous cellular tissue alone, or the deeper layers of tissue beneath the muscles and aponeuroses. The source of infection is not always apparent, the axilla and the upper extremity are regarded as the more common sites of the original infection. The cellulitis may spread over the greater part of one side of the chest, may spread downwards into the abdominal wall or upwards to the shoulder and neck. The pleura and lung may become involved in the infective process.

The disease is to be treated on the same lines as acute cellulitis in other regions, *e.g.* the neck, limbs, pelvis.

Circumscribed acute abscess of the chest-wall is met with in relation to acute osteomyelitis of the ribs, especially that form which follows upon typhoid fever.

2 COLD ABSCESS OF THE CHEST-WALL.—The common cold abscess of the chest-wall originates in tuberculous disease of a rib or costal cartilage or of the pleura. Less frequently they result from tuberculous disease of the sternum or from the extension of a spinal abscess along an intercostal space. In relation to the ribs and costal cartilages, the abscess may develop on their external surface or on their pleural aspect, or there may be an accumulation of pus on both aspects, communicating with each other across the intercostal space. The channel of communication is often very narrow, and may easily escape detection. While commonly met with in childhood and youth, they may, like tuberculous abscesses elsewhere, be met with at any period of life. They are usually situated on the lateral or antero-lateral aspects of the chest-wall. The clinical features are the same as those of tuberculous abscess in other situations. They have been known to exhibit pulsations transmitted from the heart. When left to themselves they usually make their way to the skin surface, and sooner or later rupture and give rise to a discharging sinus or sinuses. The most satisfactory treatment is to lay the abscess cavity freely open, its walls are then dealt with on general principles, any recess or channel communicating with the cavity must be explored, careful search is necessary to discover the existence of an abscess cavity on the pleural aspect of the ribs or costal cartilages, portions of the latter may require to be removed in order to deal with such a cavity when it is discovered, all diseased bone or cartilage is to be removed. The wound is then closed with sutures, or stuffed with gauze.

3 TERTIARY SYPHILIS is met with in the sternum in the form of gumma or cold abscess, or of a sinus leading down to carious bone. Its clinical features and treatment are the same as in syphilitic disease of other bones.

4 TUMOURS OF THE CHEST-WALL may be divided into those of the soft parts and those growing from the bones.

Tumours of the soft parts include sebaceous cysts, angiomata, lipomata, molluscum fibrosum, sarcoma, etc.

Tumours originating in the bones include chondromata, osteomata, and various forms of sarcomata. The latter may originate in connection with the sternum or with the ribs, they may give rise to tumours of enormous size, they may project upon the pleural aspect and press upon the lung, the parietal pleura is often involved in the new growth. The removal of a malignant tumour of the chest-wall is always a formidable operation, as it usually

entails the removal of a portion of the parietal pleura in addition to several ribs, and exposure of the lung. The skin over the tumour should be reflected in the shape of a large flap, so that it may be possible to hermetically close the pleural cavity at the end of the operation. Should the lung have collapsed during its performance, it may be confidently expected that it will re-expand and resume its functions. While the immediate result of even very formidable operations may be remarkably successful, the patient usually succumbs to recrudescence of the sarcoma at a later period.

*Secondary cancer of the sternum and of the ribs* is common enough in the advanced stages of cancer of the breast, it is not amenable to surgical interference.

#### CUTANEOUS AFFECTIONS OF THE CHEST-WALL

The skin of the chest-wall is frequently implicated in the different cutaneous affections. It is the chest that the physician first examines for the eruption of scarlet fever. The rose spots of typhoid fever, while appearing first on the abdomen, if numerous, will also be visible on the chest, the other exanthemata showing likewise their characteristic eruption. The macula syphilide appears upon the chest as an isolated blotch varying from the size of a linseed to about the size of the finger nail, the colour ranging from pink to bluish red or even a brownish tint, not disappearing entirely on pressure. Scattered throughout the blotches, or following upon them the papular syphilide may be seen somewhat elevated above the surface, varying in size up to a pea, and of a reddish dusky colour. Herpes zoster, the vesicular eruption found on the chest along the distribution of an intercostal nerve, pityriasis in its various forms, chlorasma, and other skin affections, will be found fully discussed under their different sections.

#### PAIN IN THE CHEST

The descriptive localisation of pain given by different sufferers is frequently very misleading, it is well, therefore, in every case to ask the patient to place the hand on the part implicated. Pain complained of in the chest may have an origin outside the thorax and its contents, as in *Cardialgia* from chronic gastritis or hyperchloridia, the pain resulting from irritation of sensory fibres in the cardiac end of the stomach and the lower end of the œsophagus, either by the products of fermentation and putrefaction (acetic acid, fatty acid, lactic acid) or by the hypersecretion of hydrochloric acid. The pain is then localised at about the junction of the seventh costal cartilage with the sternum, and in the back in the left interscapular region. The pain is of a burning character, and is fre-

quently relieved by vomiting. In *hysteria*, especially where there is ovarian irritation, mammary and infra-mammary pain and tenderness is common. The patient shrinks from the slightest touch, especially if her attention be directed to the part, frequently, but by no means always, if the attention be diverted, the part can be handled without producing pain or shrinking. In these cases the mammary tenderness will be accompanied by pain on pressure over the ovaries, increasing at the menstrual period, and frequently with hyperæsthetic areas in the spinal region. Pain in the chest may be of muscular origin. *Myalgia*, the so-called muscular rheumatism, when attacking the intercostal muscles and fasciæ is usually spoken of as *Pleuralgia*. Its connection with rheumatism is not always clear, it may result from strain, fatigue, cold, or other injury to the muscle, and from constitutional causes. The pain may be intense, breathing deeply, coughing, or manipulation of the muscle causing marked increase of the suffering. It is usually localised to one group of muscles. There may be slight pyrexia, but the constitutional symptoms are not marked. A good example of the affection is met after severe coughing, when myalgia may develop in the lower intercostal spaces. The pain of myalgia is frequently relieved by resting the affected muscle, a fact which the sufferer soon discovers, assuming an attitude which will throw the affected group of muscles as far as possible out of action. Pain in the chest may be the result of implication of the intercostal nerves. *Intercostal neuralgia* gives a pain more or less continuous with acute exacerbations, following the line of the intercostal nerve, with tender points near the spine, the mid-axillary line, and near the middle line in front. The pain may be increased by movement, deep breathing, coughing, etc., but is not, as a rule, so much affected by these as in other painful chest affections. Occasionally *intercostal neuritis* may develop, there is more or less severe pain along the line of the affected nerve markedly aggravated by pressure, with possibly other signs of neuritis, and frequently the further development of Herpes zoster. A symmetrical pain along the course of a group of intercostal nerves may develop in the course of affections of the spinal cord and meninges where there is implication of the sensory roots, the most typical example of the condition being found in the so-called girdle pains of tabes dorsalis.

While pain in the chest may result from affections of the chest-wall it is frequently a manifestation of grave disorder of the thoracic organs. The heart and pericardium are not normally endowed with any great degree of sensibility, but in disease the sensibility may be greatly exaggerated. In *Pericarditis* the subjective sensory disturbances vary much

There may be merely a feeling of discomfort and uneasiness in the precordial region, or the pain may be a marked symptom. The pain is usually in the precordial region, but may be localised in the epigastrium. In addition to pain there is hyperæsthesia (see "Pericarditis").

In the affections of the heart, *pain* is an extremely variable quantity. It may be an aching, the protest of an overworked muscle, the intense discomfort and distress resulting from dyspnoea, or the fully developed attack of angina pectoris. The difference is one of degree, not of kind. The special diagnostic features will be described in the article "Heart."

Pain of *pleural* origin has also to be differentiated. Here the manner of onset, site, and character of the pain, with the physical signs, readily suffice to distinguish it. Apart from the pain of acute pleurisy, there may be pain of a dull aching character, and persisting for a lengthened period, due to pleural thickening and adhesions, the result of previous inflammation.

Pain from *aneurysm of the aorta* may result from pressure, the tumour implicating a sensory nerve. Much of the pain, however, may be of the nature of a *referred sensation*. The pain from direct pressure will vary with the position of the aneurysmal tumour and the structures implicated.

In *aortitis* pain may be present in the first, second, and third right intercostal spaces. In *mediastinal growths* pain may be severe, and is due as in aneurysm to pressure upon sensory nerves.

Pain in affections of the *lung*, such as pneumonia, phthisis, etc., results, as a rule, from implication of the pleura; a referred pain may, however, be noticed along the course of the intercostal nerves. Pain in inflammatory affections of the chest-wall, mamma, etc., belongs to the domain of surgery.

The diagnosis of *intrathoracic pain* depends upon the diagnosis of the condition from which it results; and to this end the condition of pleura, cardiac muscle, aorta, and pericardium must be each in turn carefully investigated (see "Pericarditis").

#### DISORDERS OF THE CIRCULATION IN THE CHEST-WALL

Considerable distension of the veins may be noticed in cases where tricuspid regurgitation is present. In obstruction to the portal circulation as in cirrhosis of the liver, there is a marked distension of the superficial veins in the lower thoracic region along with distension of the superficial abdominal veins; the anastomotic branches of the inferior mammary and epigastric veins relieving the portal system in part, become then enlarged and visible. An enormous enlargement of the veins of the thoracic wall forming an intricate network of distended vessels may be visible when there is

obstruction to the return of blood from the chest-wall to the heart. This interference with the circulation may result from the pressure of a tumour (new growth or enlarged glands) on one or all of the great venous trunks or from thrombosis. If the superior vena cava be implicated, the venous distension will be bilateral; if one of the innominate subclavian or axillary trunks, the distension will be unilateral. It must not be forgotten, however, that in nursing women there is usually a considerable dilatation of the superficial thoracic veins during the period of lactation; the same may be seen during pregnancy and menstruation.

*Edema of the Chest-Wall.*—(Edema of the chest-wall may be present as part of a general oedema in heart and kidney disorders. There is then considerable oedema of legs, thighs, back, and passing up the posterior aspect of the chest-wall, usually symmetrically, but possibly more marked on one or other side, if the patient has been lying in a lateral position. Oedema localised to the chest-wall, arm, and head may be present when there is obstruction to the venous return, as in mediastinal tumour. A localised oedema of some diagnostic significance may occur in the chest-wall in purulent exudation into the pleura. Thus the writer has seen a localised oedema over the tenth and eleventh ribs over an encysted empyema, the oedema being of considerable diagnostic value in the absence of the usual signs of effusion into the pleura. Angio-neurotic oedema is rare in the chest, but may occur. Oedema of the chest is not to be mistaken for myxœdematous swelling of the skin and subcutaneous tissues with their characteristic dry, harsh, and pale appearance, swollen, thickened, and brawny, and not pitting on pressure.

#### EMPHYSEMA OF THE CHEST-WALL

Subcutaneous emphysema of the chest-wall, a somewhat uncommon occurrence, may result from the passage of air into the tissues, or from the development of gas within the tissues. The latter is the result of bacterial action, and does not here concern us. The passage of air into the tissues may occur under various conditions. In the lung of the child interlobular emphysema is a possible and by no means uncommon accident as the result of whooping-cough or capillary bronchitis, where the cough is frequent, violent, and paroxysmal. When the condition is marked, the air in the interlobular connective tissue may pass along the connective tissue surrounding the bronchi to the root of the lung, and thence diffuse into the mediastinum, neck, trunk, and the general surface of the body. In the adult, the lobules of the lung not being separated by distinct intervals of connective tissue, such an accident is impossible. In the adult the air may enter the mediastinum and subcutaneous tissues through a lesion of some



portion of the respiratory or alimentary tract. Ulceration of the larynx, trachea, bronchi, or wound of the pleura and lung may result in emphysema of the chest-wall. Given the formation of a cavity in the lung, the surfaces of the pleura having become adherent, the ulcerative process may pass through the parietal pleura, and a sudden effort of coughing produce emphysema of the chest-wall. Thus the air may pass directly from the respiratory tract into the chest-wall or by way of the mediastinum. From the alimentary canal the air may pass into the mediastinum through ulceration of the oesophagus by a malignant growth, or into the abdominal and chest wall from the stomach or intestine, the viscera becoming adherent to the abdominal wall and the ulcerative process subsequently invading the connective tissue.

In emphysema the skin is pale and elevated above its surroundings. On palpation the part is found to be very soft and yielding, quite unlike the brawny feeling of oedema. There is no pitting on pressure, the indentation made at once disappearing, and on handling the part there is palpable and at times audible crackling. When the air has passed by way of the mediastinum the swelling appears first at the root of the neck, passing up the neck and down over the chest-wall, and so advancing, obliterating the normal outline. When the air under the skin remains aseptic, absorption may take place; if, however, decomposition occurs, very grave complications result. In any case the accident is a very serious one, and, giving rise to distressing dyspnoea, is frequently the final development in an otherwise serious condition.

#### TUMOURS OF THE CHEST-WALL

These belong mainly to the domain of surgery. There are, however, certain points that must be borne in mind and carefully investigated in cases of obvious localised swellings or new growths. These may be summarised as follows:—

(i.) A careful examination of the condition of the aorta, anteriorly and posteriorly, with the view of eliminating aneurysm.

(ii.) The question of a mediastinal new growth should be considered.

(iii.) If the tumour be a new growth, is it primary or secondary to disease of the viscera, e.g. pleura, liver, etc.

(iv.) If there are obvious indications of the presence of fluid, is the condition a superficial localised one, or has it connection with a serous cavity.

#### AFFECTIONS OF THE MUSCLES OF CHEST

Myositis, or inflammation of muscle, is rare as a primary disease, but is more common as the secondary result of a septic process; thus the inflammation may go on to suppuration. *Rheumatic myositis* or myalgia is common in the

intercostal muscles and fascia. Its differential diagnosis will be found discussed under "Chest Pain." *Trichinosis* is an uncommon disease in Great Britain, but is more common in Germany, and affects the muscles of the chest-wall along with the other muscles of the body. The affection is due to the presence of an embryo parasite in the muscles (see "*Trichina Spinalis*"). Along with general disturbance and alimentary symptoms there are intense muscular pain, swelling, tenderness, and oedema. If the respiratory muscles become involved, there is dyspnoea. In atrophic conditions of the muscles of the chest-wall fibrillary contractions may be visible, slight momentary contractions, visible, painless, but perceptible to the patient, and best brought out by pressing the muscle or drawing the finger across the surface. Atrophy of the muscles may arise from various causes. It may be part of a general muscular atrophy in a wasting disease, as in phthisis; it may result from separation of the muscle from its trophic centre, as in neuritis; or from destruction of the trophic centre in the cord, as in poliomyelitis anterior acuta. The muscles around the shoulder-joint may atrophy in disease of the joint, the atrophy being usually described as reflex. Progressive muscular atrophies do not as a rule affect the muscles of the thorax.

Paralytic affections of the muscles of the chest-wall are usually the result of lesions of the central nervous system. Partial paralysis of the intercostal muscles may be noticed in hemiplegia, but the fully developed bilateral paralysis occurs in its typical form in transverse myelitis of the cervical region of the cord. There is then entire paralysis of the intercostals with loss of movement in the chest, the respiratory movements being entirely abdominal. In diphtheritic paralysis the intercostal muscles and diaphragm may be involved, rendering respiration difficult or impossible. Local paralysis of individual muscles may occur, for example the pectoralis major, from implication of the nerve trunk to the muscle. Hypertrophy of the muscles of the chest may, up to a certain point, result from over-use, as is seen in the respiratory muscles in emphysema.

#### Cheyne-Stokes' Respiration.—

A rhythmical irregularity of the respiration in which there is a period of gradually increasing and then gradually decreasing respiratory activity (with an acme or maximum of forcible inspiration and expiration), followed by a period of apnoea or cessation of all such movements. See *RESPIRATION (Cheyne-Stokes)*. See also *BRAIN, AFFECTIONS OF BLOOD-VESSELS (Occlusion of Cerebral Vessels)*; *BRAIN, TUMOURS OF (Symptoms, Localisation)*; *BRAIN, SURGERY (Compression)*; *CHEST, CLINICAL INVESTIGATION OF (Inspection, Respiratory Rhythm)*; *HEART, MYOCARDIUM AND ENDOCARDIUM (Affections of Myocardium, Symp-*

**tomatology, Dyspnea**); LUNGS, VASCULAR DISORDERS (*Pulmonary Embolism, Clinical Features*); MENINGES OF THE CEREBRUM (*Purulent Meningitis, Symptoms*); MENINGITIS, TUBERCULOUS (*Symptoms*); URÆMIA (*Symptoms, Respiratory System*).

**Chian Turpentine.**—An oleo-resin obtained from *Pistacia Terebinthus*, once used (with hopefulness) as a possible remedy for cancer of the uterus.

**Chianciano.** See BALNEOLOGY (*Italy, Calcareous Waters*).

**Chiari's Salpingitis.** See FALLOPIAN TUBES (*Tumours*).

**Chiasma.**—A decussation or crossing, especially that of the optic nerves. See PHYSIOLOGY, SENSES (*Vision*); RETINA AND OPTIC NERVE (*Affections of Optic Nerve, Symptoms, etc.*).

**Chick - Pea.** See TOXICOLOGY (*Food-Staffs, Vegetable, Lathyrism*).

**Chicken.** See INVALID FEEDING (*Meats*).

**Chicken-Breast.** See RICKETS (*Clinical Features, Chest*).

**Chicken-Pox.** See VARICELLA. See also INFECTION (*Rules for Prevention*); NEPHRITIS (*Etiology*); SKIN DISEASES OF THE TROPICS (*Infective*).

**Chigger.**—The chigger or *Pulex penetrans*, a sand-flea common in tropical Africa and China. See SKIN DISEASES OF THE TROPICS (*Caused by animal parasites*).

**Chilblains.** See ERYTHEMA (*Erythema pernio*). See also ALCOHOL (*External uses*); GANGRENE (*Frost-bite*); GOUT (*Irregular, Circulatory System*); LUPUS ERYTHEMATOSUS (*Diagnosis*).

**Child.** See CHILDREN.

**Childbed.** See PUERPERIUM.

**Childbirth.** See LABOUR.

**Child-Growing.** See LARYNX, LARYNGISMUS STRIDULUS.

**Childhood.** See CHILDREN; ADOLESCENT INSANITY; etc.

**Children.** See CHILDREN, DEVELOPMENT AND CLINICAL EXAMINATION; ANÆSTHESIA, CHLOROFORM (*Points in Special Cases, Children*); ANÆSTHESIA, ETHER; ASCITES (*Causation, in Children*); BLADDER, INJURIES AND DISEASES (*Calculus Vesicæ, Lithotomy*); DIABETES MELITUS (*Etiology*); GASTRO-INTESTINAL DISORDERS OF INFANCY; MESENTERIC GLANDS; NEW-BORN INFANT; PNEUMONIA, CLINICAL (*Childhood*);

PULSE (*Frequency*); RHEUMATISM IN CHILDREN; SCHOOL CHILDREN, MEDICAL EXAMINATION; SYPHILIS (*in Children*); TETANY; TUBERCULOSIS.

## Children, The Development and Clinical Examination of.

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The child differs from the adult not only in his small size, his softness, and his inability to do things, but also, very importantly, in the fact that, if he is healthy, he is constantly growing in bulk, in endurance, and in all sorts of capacity. In examining sick children, therefore, we have not only to face the ordinary problems of clinical medicine, but we are also continually being met with the question, Is the patient normal for his age as regards growth and development? If we cannot satisfy ourselves on this point, we are likely to miss much that is of importance in the case. In dealing with the investigation of children, therefore, we shall begin with a short account of such facts of growth and development as have important clinical bearings, and then proceed to consider the examination of the various organs and systems.

**GROWTH IN WEIGHT.**—At birth the baby weighs, on an average, about 7 lbs. (5 to 12). During the first two days there is a loss of from 8 to 10 oz., resulting partly from the passage of urine and meconium, and partly from the fact that the child does not receive enough nourishment at first to make up for the tissue waste. On the third day a gradual increase begins, the birth-weight being reached again about the tenth day of life, and after this the rise continues more or less steadily. The gain may vary considerably on different days, but the average daily increase is about  $\frac{1}{3}$ –1 oz. during the first five months, and from  $\frac{1}{3}$ – $\frac{2}{3}$  oz. during the rest of the first year.

By the end of the fourth month the baby's weight should be nearly double, and by the end of the first year about three times, its original figure. During the second year the child gains from 5 to 6 lbs., during the third, about 4½ lbs., and during the fourth, fifth, and sixth, about 4 lbs a year. Thus by the end of the sixth year the weight is about six times, and at fourteen years twelve times, as great as at birth.

The advantage, from a clinical point of view of weighing infants regularly is very great. By it we can gauge more simply and surely than in any other way the extent to which a particular diet is being assimilated. If a young child is losing weight, or even not gaining it for some weeks, this is to be regarded as an important morbid symptom, and its cause searched for.

**GROWTH IN LENGTH.**—The average length of a new-born baby is 19½ or 20 in. During the first six months he grows from 4 to 5 in., and in the second, 3 to 4 in. During the second year he gains from 3 to 5 in., during the third, 2 to 3½, and during the fourth, 2 to 3. After this the gain is rather less, and amounts to 1½ to 2 in every year. By the end of the fifth year the child has generally doubled his original length.

Severe chronic dys-peptic disturbance and any other morbid conditions which profoundly interfere with the general health are apt to lead to dwarfing. Rickets, if severe during early infancy, has often this effect. Chronic disease or defect of the brain generally interferes markedly with the growth of the body.

**DENTITION.**—(A) *The Temporary Teeth.*—The temporary or milk teeth are twenty in number. The following may be given as the usual ages at which they appear:—

- |  |               |
|--|---------------|
| (1) Lower central incisors                                   | 6 to 9 months |
| (2) Upper central and upper lateral incisors                 | 8 to 11 "     |
| (3) Lower lateral incisors, and lower and upper first molars | 12 to 15 "    |
| (4) Lower and upper canines                                  | 18 to 24 "    |
| (5) Lower and upper second molars                            | 24 to 30 "    |

The teeth normally come in pairs—a tooth on one side cutting the gum about the same time as the corresponding one on the other side. Those in the lower jaw appear a little earlier than the corresponding ones above, except in the case of the lateral incisors. The intervals between the cutting of the different groups of teeth may vary considerably in healthy children, but the order in which they appear seldom varies. Irregularity in their order, and the appearance of teeth singly, instead of in pairs, is an indication of rickets. Sometimes teething begins unusually early, and infants may even be born with one or more teeth

through the gum. Early dentition has no special clinical significance. Delayed dentition sometimes occurs without any apparent interference with the child's health. Generally, however, it is attributable to rickets, and this disease should always be suspected if a child has no teeth at ten months.

*Symptoms of Teething.*—In many cases nothing unusual is noticed in the general condition of the child while the teeth are appearing through the gum, and there is little or no local disturbance. Often, however, there are signs that the baby has pain in its jaw when the teeth are coming, and there may also be localised inflammation of the gum and greatly increased secretion of saliva. With or without these local symptoms we often find rise of temperature, restlessness, loss of sleep, and irritability. The appetite also may be lost, and there may be constipation or slight diarrhoea. Often the child loses weight. Less frequently slight temporary neuroses develop, e.g. there may be a constant winking of the eyes, or a frequently recurring cough or rapidity of breathing without any pulmonary disease.

Some children show a tendency to certain diseases at the time of teething which they do not seem to have at other times. Thus it is not very uncommon to find a child who with the appearance of each new set of teeth has an attack of diarrhoea or bronchitis which resists treatment stubbornly while the teeth are in process of appearing, but which rapidly recovers (under otherwise similar conditions) when they are through. Similarly, we see infants with eczema who have a marked relapse with each new group of teeth, and often an obstinate eruption will disappear almost spontaneously whenever all the teeth have pierced the gum.

The place of dentition as a factor in the causation of disease is a matter which has been much disputed. There can be no doubt that teething is not in itself a cause of death, and that its influence in producing and predisposing to disease has been enormously exaggerated. At the same time it seems equally certain that teething, like menstruation, pregnancy, and other natural states, is often accompanied by marked symptoms both local and reflex, and like them may produce temporarily a tendency to disease which is not present at other times. When symptoms due to teething cause alarm, this is not because they are themselves such as to threaten life, but because they are apt to lead us to suspect the presence of some serious disease. Thus an attack of acute bronchitis is probably in no respect more serious in a teething infant than in one who is not teething, but in the former there may be an increase in the rapidity of the breathing and an amount of fever present which make the case simulate one of pneumonia.

The diagnosis that the symptoms in a case are due to dentition can rarely be made with any confidence until the patient is well on the way to recovery.

**Treatment**—Lancing the gums was at one time very largely practised. Now it is not considered advisable, except occasionally when the gum is swollen, red, and tense over a coming tooth, and the child is suffering local pain or showing signs of reflex nervous disturbance. Under these circumstances it sometimes gives marked relief and can do no harm.

The general restlessness and irritability of teething children may be relieved by a few grains of antipyrin, phenacetin, or bromide. An aperient generally relieves the symptoms greatly.

(B) *The Permanent Teeth*—The permanent teeth number thirty-two. The order and usual time of their appearance is as follows—

First molars	6 years
Incisors	7 to 8 "
Bicuspid	9 to 10 "
Cannines	11 to 13 "
Second molars	12 to 15 "
Third molars (wisdom teeth)	17 to 25 "

The eruption of the permanent teeth is not a usual source of irritation either local or general in childhood, but the wisdom teeth, especially those of the lower jaw, may cause some distress when they appear.

#### DEVELOPMENT OF VARIOUS GLANDULAR ORGANS

—*The Salivary Glands and the Pancreas*—Not only is the saliva very scanty in young babies, but it is also deficient in diastatic power. After the third or fourth month its amount and its functional capacity increase, but it is only towards the end of the first year, when a number of teeth have usually appeared, that its amylolytic action becomes at all fully established. The action of the pancreatic secretion on starch is practically absent at birth, and develops, *pari passu*, with that of the saliva. The pancreatic juice is relatively active in digesting proteids and fats even in new-born infants.

*The Stomach*—At birth the stomach has its fundus only slightly developed, so that it has a tubular shape and a very small capacity. According to Holt, the average infant's stomach can contain at birth only  $1\frac{1}{2}$  oz., at three months  $1\frac{1}{2}$  oz., at six months 6 oz., and at twelve months 9 oz.

The stomach plays a less important part in the process of digestion during infancy than in later life. This is owing to the gastric juice being scantier and less powerful, and to the fact that the stomach contents are not allowed to remain very long in it. The relatively small proportion of hydrochloric acid in the gastric juice of infants probably helps to account for their characteristic susceptibility to gastro-intestinal infection.

*The Intestine*—In infants the intestine is relatively long, and its muscular wall comparatively feebly developed. This latter fact helps to explain the greater tendency to constipation and to flatulent distension in young children.

*The Thymus*—The thymus gland is a large organ at birth, weighing about half an ounce, and it grows until, by the end of the second year, it weighs  $1\frac{1}{2}$  to 2 oz., after that age it gradually diminishes. The area of dulness over the manubrium sterni which it causes must not be forgotten in examining the thorax in babies.

**DEVELOPMENT OF THE SPECIAL SENSES—Eye-sight**—Within a few weeks of birth most infants are evidently pleased by a brightly coloured object or a light. By the third month the child should show that he recognises his mother's face, and after that he soon gets to know the look of many things. He is long, however, of being able to distinguish colours. He may know red and yellow in the first twelve months, but will not probably recognise blue and green until the second or third year.

In testing the sight in an infant, we may try if he follows a lighted match or other bright object with his eyes, whether he seems to recognise his mother and to see familiar objects, such as toys or food, approaching. It should also be noticed if his pupils contract with light and, if he is more than a few months old, with accommodation. If the point of a finger is suddenly brought close to the eyes of a baby over two months old who has normal sight, it causes winking.

*Hearing*—During the first day or two of life all children are deaf, but by the second week they should be able to hear loud noises quite well. If they show no signs of doing so, it may be suspected that they are either deaf or idiotic. Although children are very early attracted and pleased by noises, they cannot usually distinguish even their own mother's voice when three months old.

*Taste and smell* are both relatively well developed within the first few days of life. If they have to be investigated in childhood, familiar articles of food generally form the best tests.

*Sensitivity to touch, temperature, and pain* are not very acute in early infancy. To be satisfactory, the examination of the various forms of sensation in infancy must be not only carefully made but frequently repeated.

**DEVELOPMENT OF THE VOLUNTARY MOTOR FUNCTIONS**—At birth the infant's actions are automatic, and it is only when some three or four months have passed that he acquires the power to execute distinctly voluntary movements, such as turning towards those he is fond of, or pushing away from him something he does not like. By noticing the age at which a child acquires the power to execute certain move-

ments, we gain information as to his muscular, and to some extent also as to his mental development.

If a finger is laid in an infant's palm it is generally tightly grasped, and if the child is over five months old it will probably also be carried towards his mouth. Should the child's fingers show no inclination to close on an object placed in his palm, it is a morbid sign suggesting usually either paralysis or great mental impairment.

A healthy, normally developed baby ought to be able to hold his head up when he is three or four months old, according to the degree of his muscular vigour. Only when he is eleven or twelve months old does he permanently acquire the capacity for sitting unsupported. Some children creep before they walk, as early perhaps as the ninth month, others much later. A strong baby generally begins to try to stand by the ninth or tenth month, and he may be able to do so by himself by the eleventh or twelfth. Some children can walk before the end of the first year, others not till they are nearly eighteen months old, fourteen or fifteen months is perhaps the average age.

Any delay in the acquisition of these ordinary muscular actions requires investigation. It may be accounted for by the weakening influence of a recent illness, or by some lesion of the bones, joints, or muscles. More commonly, however, it is found to indicate the presence either of rickets or of mental deficiency.

**DEVELOPMENT OF SPEECH**—When the infant is twelve months old he will understand a good many words, and may be able himself to use one or two with a definite meaning. During the second year his knowledge of words increases fast, and before the end of it he may have begun to use short phrases. The age, however, at which normal children learn to speak varies very greatly.

Should a child be unable to speak by the time he is three years old, the cause of this should be investigated. If he has suffered much from illness, the backwardness in speaking may be due to this only, and will in that case pass off as he regains strength. The hearing should be carefully examined, comparatively slight deafness may interfere a great deal with a child's progress in learning to speak. Perhaps the commonest cause of delayed speech is intellectual deficiency, the mental condition should, therefore, always be examined in such cases (see "Aphasia," "Deafmutism").

**ORDER AND METHOD OF CLINICAL EXAMINATION**—When our patients are little children we have, while using the ordinary methods, to shorten our examination as far as possible so as not to exhaust them, and also to avoid very carefully doing anything to cause them fright or annoyance. As a general rule, while in examining adults we proceed *system by system*,

investigating in turn the alimentary, circulatory, respiratory, and other organs, in young children we go rather *by methods*. We inspect first as much as we can without touching, then we palpate all over, then auscultate, and so on. There are, however, of course, many exceptions to this rule.

Before commencing the physical examination it is always well to make a few *preliminary inquiries*. It is important, for example, first to ascertain clearly for which of the child's ailments medical advice is sought, and how long the illness has lasted. The family history is also to be inquired into, the health of the parents and other children, the state of the mother during her pregnancy, and the nature of the labour when the child was born. The previous health, development, and feeding from birth onwards are very important, and special inquiries must be made as to any symptom of congenital syphilis, and as to the dates of occurrence of any of the infectious diseases. Full details of the nature and order of onset of the symptoms of the present illness should always, if possible, be obtained.

While these inquiries are being made, the child (who should, if possible, be seated on his mother's or nurse's knee) has time to grow accustomed to the doctor's presence. At the same time the medical man, without coming too near, may learn much to guide him in his further examination of the child. Physiognomical diagnosis plays a far greater part in infancy than it does in later life, and the physician who is practised in this art will always be at an advantage. He must not, however, trust to it for such information as can only be acquired with accuracy from the ordinary and more laborious methods of examination. The state of development and nutrition, the complexion and expression, and the form of the head and other uncovered parts are to be specially noticed, as well as any traces of rickets, syphilis, or other disease. The child's demeanour and the attitude he assumes are also very significant. The number and character of the respirations should be observed, and the nature of the cough and of the voice in speaking or crying is also worthy of notice.

Then comes *palpation*, and the pulse is generally felt first, lest its rate should be altered by fright or annoyance due to the further examination. A shy child's pulse will be easiest felt while his mother keeps his hand in hers. The abdomen and thorax are next palpated, without the child's position being changed (at first, at any rate). The hand is also passed over the chest to feel the amount of rickety bonding, if any is present, and the position and character of the heart's impulse. The consistence of the muscles and the mobility of the joints should next be examined, and the state of the ossification of the cranium and possible presence of

enlarged glands in the neck should not be overlooked.

*Auscultation* should generally be practised before percussion, as being less likely to cause annoyance, and immediate auscultation is sometimes less resented than the use of a stethoscope.

Then comes *percussion*, and lastly the mouth and tongue have to be inspected, and the gums and fauces seen, and, if necessary, felt. This is the part of the examination which is most likely to make the child cry, and therefore it is left to the end. The *temperature* may be taken at any time before or during the examination.

**THE HEAD**—In examining the head we must note its size, shape, and ossification, including the condition of the fontanelle and sutures.

*Size*—At birth the average circumference of the infant's head is 13 to 13½ inches. During the first six months it gains about 3 inches. At the end of the first year the head measures about 18 inches, at two years old about 19, at five 20 to 20½, and at ten years about 21 inches. There are, however, considerable variations in the size of the normal child's head. As a general rule, where the head is unusually large or small from causes which interfere with the health of the brain it has also a characteristic shape.

*Shape*—In rickets the cranium is square and sometimes asymmetrical, sometimes it presents a natiform or bossed appearance (see "Rickets"). The hydrocephalic cranium is large and rounded, that of microcephalus small, with a receding forehead and a pointed vertex.

*Ossification*—This is investigated by palpating the anterior fontanelle, the sutures, and the back of the head (for craniotabes).

*The Fontanelle*—The normal fontanelle (i.e. anterior fontanelle) is rhomboid in shape with not very thin edges, and its membrane is stretched somewhat tensely between those, so that its surface is about the level of the surrounding bones. It presents a slight pulsation transmitted to it from the arteries at the base of the brain, and a systolic murmur is often audible over it. As the child grows older the fontanelle gradually diminishes in size, and it is usually closed between the fifteenth and eighteenth months, or at latest before the end of the second year. It is changed in various ways in disease. If there is much *thinning of the bony edges* of the fontanelle, this indicates rickets or long-continued increase of the intracranial pressure.

*Alterations in the tension and level of the membrane* give valuable information as to the state of the cranial contents. Slight increase of tension, with bulging, indicates cerebral hyperæmia, active or passive, and is therefore met with in fever, in whooping-cough and bronchitis, and always temporarily when the child coughs or cries. Great tension with marked bulging

means considerable increase in the contents of the skull, and is found with hydrocephalus, intracranial tumour, and cerebral hæmorrhage. Abnormal depression of the membrane indicates lowering of the vital powers. It is met with in acute diarrhoea and in other exhausting conditions, which call for stimulant and supporting treatment. When an infant presents symptoms which lead one to suspect intracranial disease, a normal condition of the fontanelle is always a reassuring sign.

*Alterations in the size and date of closing of the fontanelle* are also important. Great delay in closure, so that the fontanelle is too large for the age of the child, is met with in rickets, in chronic hydrocephalus, and in cretinism. Premature closure is a valuable early sign of microcephalus.

*The Sutures*—Any gaping of the sutures or thinning of their bony margins has the same significance as enlargement of the fontanelle.

**THE NECK**—The state of the lymphatic glands in the neck should be noted as a matter of routine. If any are enlarged, the area of skin or mucous membrane connected with these must be carefully examined.

Stiffness of the neck must not be overlooked. Sometimes it is due to muscular rheumatism, sometimes to cervical caries. It is sometimes a symptom of basal meningitis, or some other intracranial disease.

**THE BACK**—In addition to the characteristic curvatures due to Pott's disease and rickets, we have to look out for the apparent kyphosis which is due to paralysis of the back muscles from any cause. We have also to remember that inability to hold the back straight is a common symptom of idiocy. Any lack of suppleness and any tenderness on free movement of the spine should arouse suspicion of tuberculous spine-disease.

**THE LIMBS**—The state of the circulation in the extremities, the development of the muscles, the configuration of the bones, and the size and movements of the joints have all to be examined. Pain on movement of one or more limbs may be an early and important sign of infantile scurvy, or of syphilitic epiphysitis, or may be due to a subperiosteal fracture. Clubbing of the finger-ends may throw considerable light on the nature of an obscure heart or lung case, and the presence of tuberculous or syphilitic disease of a finger may help to elucidate the nature of a brain lesion. In children with any manifestation of rheumatism, careful search should always be made over the bony prominences of the limbs for rheumatic nodules.

**THE MOUTH AND THROAT**—*The Lips*—The appearance of the lips is mainly useful as an index of the state of the circulatory system (anæmia, cyanosis, etc.).

*The Tongue*—In young infants the tongue is more or less coated, owing to the scantiness of

the saliva. In older children we often meet with the so-called "mapped" or "geographical tongue." This condition has nothing to do with syphilis, and has, indeed, little practical importance.

**The Teeth**—In examining the teeth we have to note the stage of progress of dentition and also any abnormalities of form such as those characteristic of congenital syphilis. Flattening of the tips of the more prominent teeth in both jaws indicates that the patient grinds his teeth.

**The Gums**—Various forms of stomatitis (*q v*) exert their most characteristic effects on the gums. Sponginess of the gums should always suggest the probability of scurvy being present, although sometimes it occurs as a merely local condition.

**The Palate**—In examining the hard palate in very young infants we often see little yellowish nodules in the mucous membrane near the mesial line. These are called "epithelial pearls," being composed of degenerated epithelial cells. They have no clinical significance, and rapidly disappear.

**The Throat**—In feverish cases of any kind the examination of the throat must never be omitted. In carrying this out it is of great importance to ensure, to begin with, that the child is in such a position that, when his mouth is opened, the light will at once fall on the back of the throat. It is also necessary to take precautions against possible struggling on the part of the patient. The handle of an ordinary spoon forms the best tongue-depressor, being less likely to alarm the child than any special spatula.

Digital examination of the pharynx, fauces, and naso-pharynx is very important, especially in young infants, in whom it is often difficult to get a satisfactory view of these parts. It is especially called for when there is a possibility of retropharyngeal abscess.

**THE ABDOMEN—Inspection**—In young children the abdomen is normally more prominent than in the adult. This is due partly to the relatively large size of the liver and the narrowness of the thorax, and partly to the fact that the bowels are more readily distended by flatulence owing to the weakness of their walls and those of the abdomen. Any dilatation of the superficial veins and any redness about the umbilical region must be noticed.

Retraction or hollowing out of the abdomen is a very significant sign of cerebral disease.

**Palpation**—Tenderness on palpation of the abdomen, if at all well marked, usually indicates the presence of peritonitis or some other form of inflammation. Enlargement of the abdominal organs and tumours are generally easily felt, provided the children do not resist, and a combined rectal and abdominal examination is especially useful. In all cases of difficulty it is advisable to give chloroform.

**Percussion**—Percussion is useful in determining the state of the stomach and bowels, and in confirming the results of palpation. It is also of great value in ascertaining the presence of free fluid in the peritoneum.

**The Liver**—The lower margin of the liver can usually be made out by palpation as well as by percussion. It reaches a little below the costal margin in the right mamillary line. Diminution in size of the liver is very rare. Enlargement is common, and may be due to fatty accumulation, waxy disease, cirrhosis, passive congestion, and various other causes.

**The Spleen**—The spleen is best investigated in children by palpation. To examine it you stand on the child's right side, and, placing the right hand on the left side of the abdomen with the first two fingers over the left hypochondrium, press gently inwards and upwards. In some cases where the spleen is normal in size, and always when it is enlarged, its rounded edge will be felt as a soft and readily movable body. If the lower edge is distinctly below the level of the ribs the organ may be regarded as abnormally large. Sometimes it extends right down into the pelvis. Enlargement of the spleen is common, and may be due to syphilis, leucæmia, pseudo-leucæmia, typhoid fever, tuberculosis, hepatic cirrhosis, and other causes.

**The Mesenteric Glands**—Tuberculosis of the mesenteric glands is an extremely common condition from a pathologist's point of view. Clinically, however, it is only occasionally that we are able to make sure of its presence during life, as in many of those cases in which the glands are most enlarged there are other changes in the abdominal cavity which render them difficult of palpation.

**EXAMINATION OF THE FÆCES**—The *meconium* which the infant passes during the first three or four days is of a dark greenish-brown colour, of a viscid semi-solid consistence, slightly acid in reaction, and without odour. It is sterile at birth, but within a few hours micro-organisms find their way into it through the anus. After four or five days the motions cease to contain meconium, and assume the characters of normal infantile feces.

**Normal Fæces**—In a healthy breast-fed baby the motions are from two to four in number daily during the first month or two, and usually two, but sometimes only one, daily after that. They are of an orange-yellow colour, and of a uniform semi-solid consistence. Their reaction is acid, and they have a slightly sour but not offensive odour. The stools of a hand-fed infant are similar, so long as his food resembles breast-milk in composition and is well digested.

The motions of a healthy child may vary in number, consistence, colour, reaction, and odour according to the character of the food given him. The number of the motions is increased and the consistence lessened by increase of

cream in the food, and they become fewer and more solid if the cream is lessened or the casein increased. The colour depends for its shade largely on the percentage of fat, being lighter if this is small in amount. The darkening of the faeces by bismuth and iron is to be remembered. The reaction may become alkaline from changes taking place in the incompletely digested proteids. The addition of beef-tea or raw-meat juice to the diet of a milk-fed baby causes the faeces to acquire an offensive odour.

By the end of the first dentition the motions have assumed a brownish colour, and are usually formed.

**Abnormal Faeces**—The appearance, reaction, and other characters of the motions may be greatly changed by disease (see "Gastro-Intestinal Disorders of Infancy"). When a large amount of mucus is recognisable by the naked eye in the motions, it generally indicates disease of the large intestine.

**Pus** is found in the stools in cases of ulceration of the bowel, and also in severe catarrh.

**Blood** is passed under a great variety of conditions. When pure blood comes in any quantity from a child's bowel, apart from diarrhoea or ulceration, it generally comes from a rectal polypus. In young infants the presence of a small amount of blood in the motions is often due to an anal fissure. Copious hæmorrhage from the stomach and bowel in new-born children (melæna neonatorum) is fortunately rare, but it is comparatively common to meet with cases of spurious melæna in which blood which has come from the nose or elsewhere has been swallowed and has been passed with the motions.

Fragments of membrane are found in the stools in dysentery and in croupous enteritis. **Worms** and their ova are often present, and foreign bodies such as earth, sand, stones, etc., may be found in the case of children who suffer from pica.

**THE CIRCULATORY SYSTEM**—*The Pulse*—The following may be given as the average pulse-rate in healthy children at different ages during sleep or perfect quiet—At birth, 140 to 120 per minute, 6 to 12 months, 115 to 105 per minute, 2 to 6 years, 105 to 90 per minute, 11 to 14 years, 85 to 75 per minute. Mental emotion and bodily exercise may quicken the pulse as much as twenty or thirty beats in the minute, hence the great importance of counting it when the child is undisturbed. While the pulse-rate itself is often of little significance, its ratio to the respiration-rate is always important.

Irregularity of the pulse is not uncommon in children, and is generally of little importance. If, however, the pulse is slow as well as irregular, it is worthy of careful attention. Such a condition of pulse sometimes occurs after acute illness without any serious significance, but it

is also a common early symptom of intracranial disease.

**The Heart**—The heart lies more horizontally in infancy than in later life, consequently the apex-beat in children under four years is generally outside the nipple in the fourth intercostal space. The area of the deep, as well as that of the superficial cardiac dullness is relatively large in childhood. The normal heart-sounds in little children are peculiar in certain ways. The first sound is louder than the second in all the areas. The pulmonary second sound over the base is normally louder than the aortic. It is not to be regarded as accentuated unless it is found to be constantly louder than the first sounds over the base, when the child is at rest. Owing to the greater conductivity of the tissues the heart-sounds are heard more distinctly all over the thorax in young children than in adults.

When heart-murmurs are found they may be functional in character, or due to congenital malformation or to the result of endocarditis. Functional heart-murmurs are very rare in young children, those due to congenital malformations are, of course, much commoner than in later life. When valvular disease is present it is always of importance to ascertain, if possible, whether or not it is of rheumatic origin. For this purpose inquiry must be made as to former rheumatic manifestations, and the patient examined for present signs of the disease, especially for rheumatic nodules.

**RESPIRATORY SYSTEM**—*Inspection*—*Form of the Chest*—In the normal infant the chest is more cylindrical than in the adult, and its section is consequently more nearly circular in outline. Its shape is readily altered by anything that tends to soften the already soft chest-wall or to interfere with the free expansion of one or both lungs. The commonest alteration which we meet with is the deformity characteristic of rickets, but we also find various degrees of pigeon-breast, and occasionally, as in adults, unilateral retraction or distension, due to pulmonary or pleural disease or to spinal curvature.

Noticeable enlargement of the superficial veins over the upper part of the chest is a common symptom of enlarged bronchial glands.

*Movements of the Chest*—(a) *Their Character*—The respiratory movements of the chest are slight in infants, because the type of respiration in them is almost entirely abdominal. Indrawing of the epigastrium and adjacent parts on inspiration indicates, with a normal chest, that sufficient air is not entering the lungs, and is an important sign. In broncho-pneumonia it helps us to gauge the extent to which the lung is affected, while in croup its presence to any marked degree is one of the main indications for immediate surgical assistance.

If the chest is abnormally collapsible from rickets, this symptom may be present to a con-



siderable degree without indicating any serious risk.

(b) *Rate of the Respiration*—In young infants the rate of breathing is very variable, and it is difficult, therefore, to estimate it correctly. At birth the number of respirations per minute varies from 32 to 50, and during the first year from 25 to 35. During the second, third, and fourth years it is about 25 per minute. It varies, like the pulse, with the temperature and with the mental state.

Generally the important point to determine is not merely the rate of the breathing, but the ratio between that and the pulse-rate. The pulse-respiration ratio should in health be 1 to  $3\frac{1}{2}$  or 4, and any great disturbance of these proportions is of clinical significance. Increased respiration-rate with dyspnoea signifies pulmonary disease. Rapid breathing without dyspnoea may have the same significance, or it may arise from abdominal distension or rickety deformity of the chest. It is also met with, and sometimes to a marked extent, in the irritable states which sometimes accompany teething and lithæmia.

*Extra-auscultation*—Before and during the physical examination of the child's chest it is very important to listen carefully to his breathing, his cry, and his cough.

(1) *The Breathing*—*Snuffling breathing* may be due to ordinary catarrh. When, however, it persists for a long period, or occurs apart from other catarrhal symptoms, it always suggests the presence of syphilis.

*Snoring* during sleep, with noisy breathing while awake, and a nasal tone of voice, commonly indicate the presence of enlarged tonsils or adenoids, but may be due to diphtheritic paralysis of the palate. *Noisy breathing with dyspnoea*, chiefly during inspiration, and a cry which is nasal but not generally hoarse, is characteristic of retropharyngeal abscess.

Deep *sighing* may mean very little, but it is one of the symptoms met with in the prodromal stage of tuberculous meningitis.

*Laryngeal or stridulous breathing* in young infants is most frequently due to congenital laryngeal stridor, but may be caused by laryngitis or enlarged bronchial glands. In older children it often indicates the presence of true or false croup, or of some other form of laryngeal obstruction such as polyp.

*Bronchial wheezing* is often readily audible in bronchitis of the larger tubes as well as in asthma.

(2) *The Cry*—From the loudness of an infant's cry we can gauge his strength to a certain extent. If he cries loud and long we may be almost sure that he has no serious acute condition of his respiratory passages. A hoarse laryngeal cry is generally a symptom of congenital syphilis in young infants.

(3) *The Cough*—The cough is *loud and*

*clanging* at the beginning of an attack of croup, and husky and stridulous at a later stage. In bronchitis it is often *deep and harsh*. In pneumonia, with accompanying pleurisy, it is *suppressed and painful*. If a child coughs loudly without wincing, you may be sure that he has not got acute pleurisy. A loud, noisy cough at night and in the morning is characteristic of a catarrhal condition of the throat with or without dyspepsia. If the cough has a distinctly *paroxysmal* character, this is always suggestive of whooping-cough, especially if it is worse at night, and sometimes ends in vomiting. Markedly paroxysmal coughs, however, are also met with in empyema, in enlargement of the bronchial glands, and in some cases of simple bronchitis.

*The Sputum*—It is only when children are between five and seven years old that they begin to expectorate naturally. Before that age they swallow the sputum.

Real hæmoptysis is rare in childhood. In phthisis it only occurs as a late symptom. It is most frequently met with in whooping-cough.

**PHYSICAL EXAMINATION OF CHEST.—Palpation.**—In examining the lungs, one of the first things to do is to ascertain the position of the *heart's apex-beat*. If it is displaced, this throws important light on the state of the lungs.

*Vocal fremitus* is often difficult to obtain in children, owing to the quality of the child's voice and his unwillingness to speak loud. In infants it is often got very satisfactorily during crying.

*Auscultation*—Little children often hold their breath when you try to auscultate them. This is annoying, but at least it proves that there is no serious lung-disease present. Loud crying does not interfere with auscultation very much. The long breaths which it necessitates make any accompaniments to the breath-sounds more clearly heard, and the pauses between the cries usually allow time to hear the heart-sounds during one or two cardiac revolutions. Whimpering and sobbing are much more troublesome.

The *breath sounds* in young babies are naturally very weak, owing to the feeble, shallow nature of their breathing. As the children grow older they get gradually louder, and by about the age of six months they have acquired the peculiar harsh character known as *rales*.

If on auscultating a child's chest the breath-sounds are found to differ in loudness on the two sides, the side with the weaker breathing is almost always the abnormal one. *Abnormal weakness* of the breathing is met with in the early stages of pneumonia, in pleuritic effusion, in collapse of the lung, and in pneumothorax.

*Tubular breathing* is more often met with in pleurisy with effusion in children than in adults, and sometimes leads to a mistaken diagnosis of consolidation.

**Friction** is sometimes difficult to make sure of in young babies, and we have to depend on the catch in the breath and the evident pain accompanying it to confirm our diagnosis. A peculiar *expiratory rhythm* of the breathing is very characteristic of childhood. In this there is a loud, long expiration followed immediately by a short inspiration, then a pause, then the loud expiration again, and so on. The pause occurs, therefore, after inspiration, instead of, as normally happens, after expiration. This type of breathing is often noticed in normal children—especially when they are frightened. It is seen markedly in pneumonia, and in it the expirations have frequently a grunting character.

**Vocal resonance** can generally be obtained best in young children when they are crying.

**Percussion**—In children a light stroke should always be used in percussion. This is advisable, not only because it is less apt to annoy the child, but also because it is more likely to lead to accurate results. Strong percussion over a part often misleads by bringing out dullness or resonance from underlying organs.

It is essential to see that the patient is *sitting straight*. Even a slight twist of the spine may give rise to distinct differences of percussion-note on the two sides of the chest. We must also beware of the differences due to abnormal curves of the ribs. Too much importance must not be attached to small areas of partial dullness in a child's chest, but at the same time it is to be remembered that the presence of fluid in the pleura often causes less absolute dullness in children than might be expected.

A well-marked *cracked-pot sound* can often be obtained in little children who have perfectly normal lungs—especially when they are crying.

**URINARY SYSTEM**—In young children the bladder is higher than in later life. This must always be remembered in tapping the abdomen.

**Micturition**—The infant may pass water very soon after birth, but often does not do so for twelve or even twenty-four hours. During early infancy incontinence is the normal condition. Some infants have acquired a certain control over their bladder by eighteen months old, in others this comes considerably later. If, however, a child cannot retain his urine to a considerable extent, during his waking hours, by the time he has reached his third year, he may be said to suffer from incontinence.

**Incontinence of urine** may be due in girls to a local malformation, and in both sexes to conditions of general debility or to various severe organic diseases of the nervous system. In the great majority of cases, however, it is a functional neurosis (see "Urination").

**Retention of urine** is much less common. It may be the result of extreme phimosis or of an impacted calculus. It may also be due to reflex irritation from an anal fissure, or from thread-worms, or to central causes, such as meningitis.

**Dysuria** is not uncommonly due in infants, as well as in older children, to acid urine. In boys it is sometimes caused by phimosis or preputial adhesions. **Renal colic** from the passage of uric-acid crystals is occasionally met with.

**The Urine**—In infants some idea of the colour and amount of the urine passed may be obtained by examining the nappies, but, where it is possible, a proper specimen should be collected.

The following table (Holt) gives the average amounts passed at different ages—

First twenty-four hours	0 to 2 ounces
Second twenty-four hours	1 " 3 "
Three to six days	3 " 8 "
Seven days to two months	5 " 13 "
Two to six months	7 " 16 "
Six months to two years	8 " 20 "
Two to five years	16 " 26 "
Five to eight years	20 " 40 "
Eight to fourteen years	32 " 48 "

**Albuminuria** is sometimes present normally during the first ten days, owing, apparently, to the passage of uric-acid crystals. In older children its significance is the same as in adults. All cases of albuminuria should be examined for traces of recent scarlatina.

**Hæmaturia** is found in acute nephritis, diseases of the bladder, and tumour of the kidney. It is also met with in infantile scurvy, purpura, malaria, and various other conditions.

**Pyuria** occurs in cystitis and pyelitis, in both cases usually with acid urine.

**Glycosuria** is rarely found to any great degree in childhood.

**THE INTEGUMENTARY SYSTEM**—The colour of the skin as well as its state as to moisture and temperature must always be noted, and it must be examined for oedema, desquamation, and eruptions.

Cyanosis setting in during an acute illness is often of great importance as indicating failure of the heart or the degree to which the lungs are affected. It is also a characteristic symptom of various forms of blood-poisoning. When habitually present it generally indicates the presence of a congenital heart-lesion. The peculiar sallow tint of the skin in infantile scurvy, congenital syphilis, and other cachectic conditions, is often of great assistance in diagnosis.

Undue perspiration in young children is usually a sign of rickets, although it also occurs in various forms of debility and in blood-poisoning. A cold, clammy state of the extremities should draw attention to the state of the digestion and general health.

**THE TEMPERATURE**—In young children it is generally more convenient to take the temperature in the groin than in the axilla, because of the attitude they assume in sitting and the disposition of their clothes. In important cases

the rectum should be preferred. The thermometer should never be trusted in the mouth in young children.

The normal temperature shows greater daily variations in infancy than in adult life, but its average level is about the same. There is a greater tendency for the temperature to vary upwards and downwards on slight occasions.

A subnormal temperature is characteristic of atrophy. In wasted babies, therefore, a temperature of 98° or 99° F may denote the presence of fever.

*Pyrexia* is sometimes caused by emotional excitement, and sudden rises of temperature are often due to trivial causes. Continuous pyrexia, however, is always important. A high temperature occurring between the second and fifth day after birth, unaccompanied by other obvious symptoms of illness, is a sign that the infant is not getting sufficient nourishment.

**THE NERVOUS SYSTEM.**—In investigating the presence of nervous disease in children we have not only to examine for the usual physical signs, but also, especially in acute cases, to make careful inquiry for a history of various suggestive symptoms.

Thus we may ask if any change has been noticed recently in the child's disposition and temper, whether he has complained of pain in the head or elsewhere, or has had photophobia or giddiness, whether he is in the habit of screaming out suddenly or sighing heavily or grinding his teeth, whether he vomits or is constipated, and if he has had convulsions.

In looking for physical signs we must not omit to notice the physiognomy, the condition of the fontanelle and of the superficial cranial veins, and to observe if the pulse is irregular. The state of the pupils, and the presence of squint or nystagmus, of cervical rigidity or head-retraction, are also important.

Defects of ordinary sensibility are less common in children than in adults. Pain on movement of a limb is important, but is not often due to disease of the nervous system.

In investigating any apparent loss of muscular power in a child, we have, *firstly*, to decide whether it is a true paralysis resulting from the pain which movement causes, from extreme flabbiness of the muscles, or from some malformation, and, *secondly*, if a true paralysis, whether it is due to a lesion of the brain, cord, or peripheral structures, or is a so-called functional palsy such as may be met with in hysteria or as the result of peripheral irritation.

Tremor is a rare symptom in childhood, but choreiform or athetoid movements are commoner than in later life.

When it is important to ascertain the condition of the electrical reaction in a young child, it is generally advisable to give an anæsthetic.

**Chill.**—An abnormally low state of the

bodily temperature, with shivering, etc., as in the cold stage of ague (see *MALARIA*), or the chilling of the body by exposure to cold, or "catching cold" (see *PLEURIA*, *DYSENTERIA*, *Acute Pleurisy*, *Etiology*).

**Chillie Paste.** See *CAPSI*, *FRUGUS*.

**Chimneys.**—In the construction of a house the chimneys should have straight circular flues, separate from each other, and should rise 3 feet at least above the roof, in ventilation they are of the greatest use as outlets, but ought not to act as inlets for air (owls help to prevent the inlet action), their action as outlets is due largely to the warmth of the fire heating the air below, and in part to the aspirating action of winds blowing across the top. In Scots law (*Public Health (Scotland) Act, 1897*) a chimney (not that of a private dwelling house) sending out smoke in such quantity as to be injurious to health is a "general nuisance."

**Chimney-Sweep's Cancer.** See *SCROTUM* and *TESTIS*, *DISEASES OF (Epidelioma of the Scrotum)*.

**Chin-Cough.** See *WHOOPING-COUGH*.

**Chinoline.**—Chinoline or quinoline ( $C_9H_7N$ ) is an alkaloid which can be made in various ways, *eg* from quinine, aniline, or glycerine, and which is closely related to benzene and pyridine. See *ALKALOIDS (Composition and Character)*.

**Chinosol.**—A proprietary compound of oxychinoline, a yellow crystalline powder, with antiseptic properties, it is stated that 15 grains of it in a pint of water make a solution equal to 1 in 40 carbolic lotion, it belongs to the coal-tar series, is non-poisonous and non-corrosive, if used with perchloride of mercury, both are rendered inert.

**Chionablepsia.**—Snow-blindness (*Gr* χιόν, snow, and *ἀβλεψία*, blindness).

**Chirapsy.**—Friction or rubbing with the hands. See *MATERNAL IMPRESSIONS*.

**Chiretta.** See *PHARMACOLOGY*, *PRESCRIBING*.—*Chirata* is the dried plant (root, stem, leaves) *Sweetia chirata*, which has a very bitter taste, and contains chiratin ( $C_{26}H_{48}O_{19}$ , bitter principle) and opheic acid, it is a bitter, but as it contains no tannin it may be made up with non-toxic official preparations are the *Infusum Chiratae* (dose,  $\frac{1}{2}$  to 1 fl oz), the *Liquor Chiratae Concentratus* (dose,  $\frac{1}{2}$  to 1 fl dr), and the *Tinctura Chiratae* (dose,  $\frac{1}{2}$  to 1 fl dr). For *Indian Chiretta*, see *ANDROGRAPHIS*.

**Chiropract.**—A person who treats diseases of the hands and feet, but more especially (or entirely) corns and bunions.

**Chloasma.**—A pigmented condition of the skin in which yellowish or brownish patches appear, e.g. in pregnancy (*chloasma uterinum* or *gravidarum*), or in tubercle (*aphthiscorum*), or from the sun's rays (*C. caloricum*), or from the application of blisters (*C. toxicum*). See LUNG, TUBERCULOSIS (Complications, Integumentary), PREGNANCY, PHYSIOLOGY (Changes in the Skin), SKIN, PARASITES (*Tinea Versicolor*), SKIN, PIGMENTARY AFFECTIONS, SYPHILIS (*Diagnosis*)

**Chlor.**—In various compound words *chlor* occurs, and it generally signifies that *chlorine* enters into the composition of the substance for which the word stands. In other cases it simply means green (e.g. *chlorosis*). In addition to the words separately considered *infra*, the following may be referred to here: *chloroacetamide* (formed from acetamide by substituting chlorine for hydrogen), *chloracetone* (formed from acetone by substituting chlorine for hydrogen), *chloroacetylene* (a chlorine substitution compound of acetylene), *chloralantipyrene* (a compound of chloral and antipyrene, acting as a hypnotic), *chloraloin* (a substance formed by passing chlorine gas through solution of aloin), *chloralose* (a substance formed by reaction of anhydrous chloral and glucose, and acting as a hypnotic), *chlor-alum* (impure chloride of aluminium, acting as a disinfectant), *chloramyl*, *chloraniline*, *chlorbenzene*, *chlorocaffeine*, *chlorocamphor*, *chlorocyanogen*, *chlorotone*, etc. etc

**Chloral Hydrate.** See also ANALGESICS AND ANODYNES, COLOUR VISION (*Acquired Colour-Blindness*), CONVULSIONS, INFANTILE, DRUG ERUPTIONS (*Erythematosis*), ECLAMPSIA (*Treatment*), HYPNOTICS, LABOUR, PRECIPITATE AND PROLONGED (*Pauses in the Soft Passages*), PHARMACOLOGY, PRESCRIBING, PURPURA (*Symptomatic Purpura, Toxic*), TEMPERATURE (*Depression*), TOXICOLOGY (*Organic Poisons, Chloral Hydrate*)—Chloral Hydrate consists of clear, translucent crystals, with a characteristic aromatic odour, and a bitter, pungent taste. It liquefies on heating, and is readily soluble in water, alcohol, ether, and certain oils. A thick oily liquid results when it is mixed with camphor or menthol. It is given in doses of 5-20 grs. **Preparation**—Syrupus Chloral, each fluid drachm containing 10 grs. **Dose**— $\frac{1}{2}$ -2 drs. Chloral is a pure hypnotic, causing a natural sleep, which is not followed by any unpleasant after effects. It has no analgesic action, however, and is therefore of little use alone in conditions associated with severe pain, such as neuralgia. Its administration is of most value in cases of simple sleeplessness, especially if associated with restlessness and excitement, in such cases it may with advantage be combined with bromide. On account of its depressant action it must be given with caution to patients labouring under cardiac or respiratory embarrassment. It is a strong

irritant, and may cause vomiting unless taken well diluted. If the patient is for any reason unable to swallow, it may be administered by the rectum or hypodermically. There is a strong tendency for the formation of a chloral habit, and its use as a regular sedative and hypnotic in chronic diseases is therefore contra-indicated. The following are some of the diseases for which it is recommended—mania, delirium tremens, whooping-cough, convulsions, tetanus, strychnine poisoning, severe chorea, chorea of pregnancy, asthma, eclampsia, and uræmia with nervousness and restlessness.

**Chloralamide.**—A hypnotic compound, *Chloral formamide*, given in doses of 15 to 40 grams, it is not official, it dissolves only slowly and imperfectly in water, but better in acid solutions. It is said that the proprietary preparation *chlorobrom* resembles a mixture of chloralamide and bromide of potassium. See also HEART, MYOCARDIUM, DISEASES (*Treatment, Medicinal, Sleeplessness*)

**Chloralism.**—Chronic poisoning due to the chloral habit. See TOXICOLOGY

**Chlorate of Potash.** See POTASSIUM, PREGNANCY, INTRA-UTERINE DISEASES (*Intra-Uterine Therapeutics*), PRESCRIBING

**Chloretone.**—Chloretone ( $\text{CCl}_3(\text{CH}_2)_3^1\text{OH}$ ) is a trichloro-derivative of tertiary butyl alcohol ( $\text{C}_4\text{H}_9\text{OH}$ ), obtained by slowly adding caustic potash to equal weights of chloroform and acetone, it is anæsthetic (like cocaine) and antiseptic. See NOSE, EXAMINATION, LOCAL ANÆSTHETICS

**Chloric Ether.**—*Spiritus Chloroformi*, one of the official preparations of chloroform, given in doses of 5 to 20 m, or in larger doses (20 to 40 m) if for one administration. See ANÆSTHESIA, CHLOROFORM, PRESCRIBING

**Chloride of Ethyl.** See ANÆSTHESIA, ETHYL CHLORIDE, TEETH (*Tooth Extraction*)

**Chlorides.** See DIET (*Mineral Constituents*), URINE, PATHOLOGICAL CHANGES IN (*Inorganic Constituents, Chlorides*)

**Chlorinated Lime.** See CHLORINE

**Chlorinated Soda.** See CHLORINE

**Chlorine.**—A greenish-yellow gas with intensely irritating properties. It is not official, but is represented by—

1 *Calx Chlorinata*, bleaching-powder, containing 33 per cent of available chlorine, which it gives off on exposure to air. **Preparation**—Liquor Calcis Chlorinate, a 1 in 10 solution

2 *Liquor Soda Chlorinata*, made by mixing solutions of sodium carbonate and chlorinated lime. **Dose**—10-20 m

Chlorinated lime is largely employed as a disinfectant for drains, etc., and chlorine gas was at one time a popular disinfecting agent for rooms after infectious disease. Weak chlorine-containing solutions are sometimes used as mouth washes and gargles in septic conditions of the mouth and throat. It has also been strongly recommended as an internal antiseptic in typhoid fever.

**Chlorinism.** See TOXICOLOGY (*Non-Metallic Elements, Chlorine*)

**Chlorobrom.** See CHLORALAMIDE

**Chlorodyne.** See MORPHINOMANIA (*Chlorodyne Habit*), TOXICOLOGY (*Alkaloids*).—A proprietary preparation, used as a hypnotic and anodyne, it is believed that the official *Tinctura Chloroformi et Morphine Composita* has a somewhat similar composition, the latter contains morphine hydrochloride dissolved in a mixture of chloroform, tincture of capsicum, tincture of cannabis indica, oil of peppermint, glycerine, and alcohol, with some dilute hydrocyanic acid added to it after the morphine has been dissolved in it.

**Chloroform.** See ANÆSTHESIA, GENERAL PHYSIOLOGY, ANÆSTHESIA, CHLOROFORM. See also ABDOMEN, CLINICAL EXAMINATION, ANPHYLXIA (*Causes*), ANALGESICS AND ANODYNES, ANTIPYREPTICS, CHILDREN, CLINICAL EXAMINATION OF (*Abdomen*), CONVULSIONS, INFANTILE (*Treatment*), ECLAMPSIA (*Treatment, Arrest of Fits*), LABOUR, MANAGEMENT OF (*Anæsthetics*), MORPHINOMANIA AND ALLIED DRUG HABITS (*Chloroform, self-administration*), OXYGEN, PHARMACOLOGY, PRESCRIBING, TOXICOLOGY (*Organic Poisons, Chloroform*).—Chloroform, as an anæsthetic, is dealt with fully elsewhere, but it is necessary here to add a few particulars regarding its pharmaceutical characters. Chemically, it is trichloromethane ( $\text{CHCl}_3$ ), and is a clear, colourless liquid, with a pleasant smell and taste. There are four official preparations of it: *Aqua Chloroformi* (dose,  $\frac{1}{2}$  to 2 fl oz), *Linimentum Chloroformi*, *Spiritus Chloroformi* or Chloric Ether (*qv*), and *Tinctura Chloroformi et Morphine Composita* (dose, 5 to 15 in). *Chloroformum* itself may be given internally in doses of 1 to 5 in

as shown by the formation of numerous petechiæ and ecchymoses, and suppuration occurs readily. Exophthalmos, an important diagnostic sign, is almost invariable, and becomes more marked as the disease progresses. Pain in the eyes and temporal regions is usual in the later stages. There may be some enlargement of lymphatic glands. Examination of the blood reveals a progressive anæmia, which may reach an extreme degree, and a marked leucocytosis, due to an enormous increase of lymphocytes. A few myelocytes and nucleated red corpuscles are also usually present. The diagnosis depends on the leukæmic condition of the blood, the exophthalmos, and the evidences of tumour formation especially affecting the bones of the skull. The disease is always rapidly fatal. Authorities are at variance as to the exact nature of chloroma, but it is now recognised that the tumours belong to the class of lymphosarcomata, with a tendency to rapid and extensive metastatic formation, and that they originate chiefly in connection with the periosteum of the bones of the skull and face. In spite of the similarity of the blood picture to that of lymphatic leukæmia and other points of resemblance between the two diseases, it is probable that the conditions are essentially different. The distribution of the tumour masses is very wide, and they may be found in almost every organ of the body, but the brain, spinal cord, and nerves escape. The green colouration rapidly fades when the tumours are exposed *post mortem*. The nature of the colouring matter has not yet been determined. Some regard it as being due to small clusters of fatty material scattered about the substance of the tumours, others to a chemical product allied to lipochrome, which is oxidised on exposure to light.

### Chlorosis.

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See also ANÆMIA, ANÆMIA, PERNICIOUS, BRAIN, AFFECTION OF BLOOD-VESSELS (*Thrombosis*), LEUCOCYTOSIS (*Leucopenia*), MENSTRUATION AND ITS DISORDERS (*Amenorrhœa*), SKIN, PIGMENTARY AFFECTIONS (*Chlorosis*)

**Chloroma.**—A rare disease, in which there is a widespread development of soft green-coloured tumours of a lymphosarcomatous nature, and associated with blood changes closely resembling those of lymphatic leukæmia. It occurs most frequently, but not exclusively, in children under fifteen years of age, and is more common in males than in females. The early symptoms are progressive wasting, weakness, and loss of appetite, with extreme facial pallor, which later gives place to a waxy-yellow complexion. There is also a hæmorrhagic tendency

INTRODUCTORY.—Chlorosis or green-sickness is a disease of the female sex, occurring usually for the first time between the ages of fourteen and twenty years, and having anæmia as its cardinal symptom. The anæmia is due to defective blood formation, it occurs spontaneously, or at least without any cause which is universally admitted to be sufficient, develops rapidly as a rule, and gives rise secondarily to a number of other symptoms. If the disease is not treated it does not tend to recover, but to become more

severe or chronic, and even when efficiently treated it is apt to recur. As the patient advances in years this tendency becomes less marked.

**CAUSATION AND PATHOLOGY.**—(1) *Predisposing Causes.*—So far as is known, chlorosis occurs only in the female sex, and the first attack usually falls in the years between fourteen and twenty, very seldom earlier and seldom later. Recurrences may occur at any time after the first attack, and have been met with comparatively late in life. The disease often runs in families, especially in large families, and the mothers of chlorotic girls have themselves often been affected. No other illnesses in the parents have any special relation to chlorosis in the children. There must be a predisposing weakness of a functional character in the blood-forming organs, but Vichow's belief that the cause of the disease is a general hypoplasia of the vascular system, and the kindred notion that it is due to a hypoplasia of the genital organs, are certainly erroneous. It is impossible that a disease so curable should depend on organic defects so serious.

(2) *Exciting Causes.*—Such bad hygienic conditions as want of fresh air and of light in the rooms which patients inhabit, and such faults in clothing as corsets so tight as to interfere with the proper action of the viscera, may assist in producing the disease. An improper amount of work and exercise is probably more important, and more harm is done by too much fatigue than by too little. Unwisdom in the choice of food or inability to obtain a proper quantity or quality is even more serious. The iron of the blood is normally regenerated from the iron of the food, though there is a reserve in the liver which can be drawn upon in emergency. Normally the intake of iron in the food, in men at least, just balances the output, but young women tend to prefer other foods to the non-containing proteins of a man's diet, and their intake of iron is thus often insufficient. Moreover, chlorosis is very apt to appear at the time when girls have left school and home, and have begun to work. They need a fuller diet, especially more proteid food, and often cannot afford it, are not allowed it, or will not take it. Bad hygienic conditions, dyspepsia, constipation, mental depression, all lead to loss of appetite and to a diminution in the amount of food taken. In addition to a diminished intake of iron there is in young girls an excessive output. The blood-loss of menstruation means on each occasion an output of iron equal to that ingested in the food in a week, and it must require a healthy appetite and vigorous digestion to make good the loss. Many chlorotics have at one time suffered from menorrhagia, though when the disease is fully established the majority either cease to menstruate, or do so in diminished quantity. In the chlorotic years the de-

velopment of the whole body goes on rapidly, and must throw a great strain on the blood-forming organs, and if these are imperfectly nourished, and exposed to loss as well, chlorosis may well result. Very little is known of the mechanism by which the blood-forming organs are stimulated to make good any loss, and it is possible that this may depend to a certain extent on the internal secretion of one or more organs, in the female sex possibly the genital organs. A loss of this stimulation from functional disturbance of the generative organs may endanger the normal course of blood formation.

The view that chlorosis is dependent on constipation has been abandoned. Constipation is not more common nor more severe among chlorotics than among other women, and purgatives alone will not cure the disease. Nor is there ground for believing that an abnormal amount of intestinal putrefaction is present to produce toxins which destroy the blood or hinder its formation. The ordinary evidences in the urine of increased intestinal decomposition are wanting in chlorosis, nor is there any reason to think that excessive blood destruction occurs.

(3) *Pathology.*—Chlorosis is not a fatal disease, and hence little or nothing is known of its morbid anatomy. Fatty degeneration of the heart and other viscera has been found in this as in other anæmias, the stomach may be dilated, and so may the heart. The only change which is characteristic is the condition of the blood, and the only alteration there which is absolutely constant is the diminution of hæmoglobin, not only absolutely, but also relatively to the red corpuscles. The red corpuscles in the cubic millimetre vary greatly in number. In cases which are taken into hospital they are often diminished, though very rarely below two millions. These, of course, are the severe cases. In out-patient and private practice the number is more usually about three or four millions, and not infrequently the number is normal—about 4,500,000—or above the normal. But even in the cases with a normal number of corpuscles the hæmoglobin will often be diminished to 50 per cent—a colour-index of 0.5—while when the corpuscles are unusually low the hæmoglobin also sinks. I have taken 313 cases, tabulated by Thayer, Cabot, Bramwell, and Stockman, and find the average number of red corpuscles to have been 3,800,000, while the average percentage of hæmoglobin was 40.3. This gives a colour-index of 0.47.

The blood when drawn is pale and very fluid, but coagulates rapidly, though the fibrin is not increased. Blood-plates are almost always increased in number, indeed, sometimes in stained films they seem to be almost as numerous as the red cells.

The red corpuscles are small, and when stained are pale, and show specially a much paler centre than usual. This of course corresponds with

their individual poverty in hæmoglobin. In slight cases their shape is often otherwise normal, but in severe cases poikilocytosis may be quite as marked as in pernicious anæmia, though the deformed corpuscles still show the central pallor. Nucleated red corpuscles are extremely rare, and are only found in the severest cases. Leucocytosis may occur in chlorosis from any of the causes which ordinarily produce it, but in uncomplicated cases the leucocytes tend to be scanty in number rather than increased, and to be fewer in the worst cases than in the milder ones. The percentage of lymphocytes is typically increased. The specific gravity of the blood and the dry residue are diminished parallel to the hæmoglobin, but the plasma is unaltered. If a case be watched through its development and recovery it will be found that at first the red corpuscles are normal in number, but they become small in size and the hæmoglobin is diminished. Then the number of the corpuscles begins to fall and poikilocytosis appears. As the case improves under treatment the corpuscles increase rapidly in number, but remain for a long time small and pale, and the normal number has been regained long before the corpuscles attain their normal size, and before the hæmoglobin reaches the normal.

The essence of the disease is insufficient blood formation, one might indeed define it as a lassitude of the bone marrow. The demand of the body for fresh corpuscles is complied with, but the corpuscles are small and of light weight, and the oxidation processes in the body are thus interfered with.

**SYMPTOMS**—The first complaint is usually breathlessness on exertion, then follow fatigue and palpitation. Pallor, which may be greenish in tint, does not usually appear till the blood changes are well advanced, and shows itself first in the lips and conjunctivæ, later in the skin. The patients often, indeed, have a fresh pink and white complexion, for the vascular nerves are easily excited and flushing results. There is disturbance of menstruation, usually greater the younger the patient and the more seldom she has menstruated before. Complete or partial amenorrhœa is the rule, menorrhagia is much less common. This suppression of menstruation is of course a curative endeavour. Leucorrhœa is common. The muscular power diminishes, though here there are great individual differences due rather to temperament and will-power than to bodily conformation. Headache is common, and in bad cases one meets also tinnitus, temporary blindness, and deafness, giddiness, and fainting. The last is specially apt to occur after the patient has been standing for a long time.

The amount of alimentary disturbance varies greatly. The stomach is sometimes dilated, and gastric ulcer is a not infrequent complication. Some patients retain a normal appetite, and

others tend to become fat; others lose their appetite, suffer from dyspeptic symptoms, and become thin. But even those patients who retain their appetite are very apt to be capricious in their choice of food, meat they generally dislike, while they have a special fondness for acids, such as lemons and vinegar, or for such things as chalk, starch, dry oatmeal, dry tea-leaves, or dry sage. Constipation is common. The tongue is sometimes furred and flabby, but more usually pale and clean. The pulse is generally rapid, the heart may be enlarged, usually more to the right side than the left. Coldness of the hands and feet or of the whole body, from feebleness of the peripheral circulation, is often complained of, and patients often suffer from "dead fingers" for the same reason. Thrombosis of the veins, especially of the legs, occasionally occurs, and without this there may in severe cases be some œdema of the anæles. It is doubtful whether these thromboses are due to the increase in blood plates or not. In the majority of cases they seem to be organismal in origin. The splenic dulness is often enlarged, but the organ is rarely palpable. The urine is copious, pale, and of low specific gravity.

The patients are often irritable, capricious, or obstinate. The headache may be constant, or may occur in attacks like migraine. Neuralgias of all kinds are very common, especially infra-mammary neuralgia. Optic neuritis sometimes occurs. The temperature seldom rises unless for some complication.

**Cardiac and Vascular Murmurs**—The cardiac murmurs of uncomplicated chlorosis are always systolic in time, and are heard most frequently in (1) the pulmonary area, with the point of maximum intensity in the second left interspace, close to the sternum, i.e. just over the pulmonary artery. The causation of this murmur has been much discussed. It has been ascribed to mitral regurgitation, to pressure on the pulmonary artery by the dilated left auricle, to wateriness of the blood, and to other causes. The view now most commonly held is that it is due to a want of tone in the walls of the artery, similar to that found in the heart walls, and caused by malnutrition, and that the murmur is produced by the sudden propulsion of the blood into the artery, which is relatively dilated in comparison to its orifice. The same cause would account for the murmur which is sometimes heard in (2) the aortic area, though with much less frequency. Systolic murmurs in (3) the tricuspid area and (4) the mitral area are usually heard along with the basal murmur, but sometimes alone, and in cases where they are not due simply to the conduction of very loud basal murmurs, are more serious, for they are found only in severe cases of chlorosis, and indicate that the heart wall is so enfeebled as to allow of sufficient dilatation to cause respectively tricuspid or mitral regurgitation. It is some-

times difficult to be certain in these cases whether the murmur is due entirely to chlorotic dilatation or to pre-existing valvular disease. The history and the blood examination generally help us, and the result of treatment usually removes all doubt, as chlorotic dilatation is eminently curable.

*Arterial murmurs* are sometimes heard at a distance from the heart, but not with sufficient constancy to be characteristic. The *venous murmurs* are heard most easily in the jugular veins, at the root of the neck, especially on the right side. This *bruit de diable*, or venous hum, is continuous, though it differs in intensity from various causes. Its causation is usually ascribed to the fact that, while the upper part of the vein is free, and able to collapse if ill-filled, or if it shares in the general want of tone of the vascular system, the lower part is kept distended by its attachments to the cervical fascia. In the passage of the blood from a narrower to a wider space we have one of the typical conditions for the production of a murmur, which is continuous because of the continuous flow of blood in the veins. A murmur of the same character may sometimes be heard over the eyeball, over the occipital protuberance, and elsewhere. The venous hum, though not confined to chlorosis, is very characteristic of it.

*Pulsation* is often to be seen and felt in the episternal notch, in the pulmonary area, over the area of the right ventricle, and in the epigastrium. From what has been said of the state of the heart and vessels its causation in each case will be evident.

**COURSE AND PROGNOSIS**—Cases vary very much in their rapidity of onset. In some the disease develops in a few days, but in the great majority its incipient stages extend over two or three weeks, or even over a much longer period. Generally speaking, cases with an acute commencement recover quickly, those with a chronic commencement take much longer to get well. But all cases, acute or chronic, show a marked tendency to recurrence. This is largely to be explained by the fact that it is very difficult to persuade chlorotics to persevere with treatment until they have thoroughly regained their health. They are accustomed to a condition of health which is short of robustness, and there is often so marked an improvement after a short course of iron that they are satisfied with it, and drift out of observation. I have seen patients with a hæmoglobin percentage of 50 declare themselves quite recovered, and resent the idea of further treatment. The result is a recurrence in a short time, and these cases often become quite chronic, for relapses do not yield so well to iron as primary attacks. On the other hand, patients who will submit to treatment usually recover completely, and are not nearly so liable to relapses as imperfectly recovered cases. It is, of course, of great importance to impress upon

patients the necessity of complete recovery, for patients suffering from chlorosis are in a poor condition to resist acute intercurrent diseases; they are liable to the development of gastric ulcer, and if the disease is allowed to become chronic, though the anemia may pass off in after years, it leaves its mark in a state of weakened vitality, and often leads to a condition of chronic invalidism.

**DIAGNOSIS**—The points on which stress should be laid are—the sex and age, the apparently causeless development of the disease, the history and general appearance of the patient, the character of the anemia on examination of the blood, especially the disproportionate diminution of hæmoglobin, and the success of treatment with iron. Difficulty may arise in cases where one or other set of symptoms of the disease is unusually prominent, the gastric and cardiac symptoms, and those associated with the generative organs, being most likely to cause error, and it is of course not unusual to find chlorosis in patients who are suffering from organic heart disease, from tuberculosis, or other chronic maladies. From other conditions causing anemia the differential diagnosis should be made on the following lines—

(1) *Pernicious Anæmia* (see "Anæmia, Pernicious")—It is comparatively seldom that it is necessary to make this diagnosis, as it is very rarely that the anemia in chlorosis is so grave as to give rise to the suspicion of the other disease.

(2) *Leucocythæmia*—The examination of the blood at once removes doubt.

(3) *Anæmia from Intestinal Parasites*—The type of anemia is rather that of pernicious anemia. Where the blood examination leaves any doubt the feces should be examined for the eggs of the parasites.

(4) *Anæmia from Malignant Disease*—The type of anemia is very often chlorotic in malignant disease, but though, of course, malignant disease is not specially common in young girls without causing definite symptoms, it may occur, and sometimes does give rise to difficulty. Leucocytosis is much more common in malignant disease, nucleated red corpuscles are more common and more numerous than in chlorosis, and the effect of treatment usually clears up the difficulty.

(5) *Tuberculosis*—Nothing is more common than to find early cases of phthisis and other forms of tuberculosis taken for chlorosis, because the lungs, etc., have not been examined with sufficient care. The blood examination gives very similar results, and careful investigation of the history, repeated examination of the sputum, lungs, glands, etc., must be made. The temperature is not always helpful, for early tubercle does not always cause fever, and in chlorosis the temperature is sometimes raised. In very difficult or important cases it might be



possible to make the diagnosis by the injection of tuberculin, when reaction or its absence would make the diagnosis easy. It is to be remembered that tubercular tumour of the brain or meningitis may cause anaemia.

(6) *Simple anaemia* from bad hygiene, want of food, of sleep, of light, of fresh air, from overwork, etc.—In these cases the hæmoglobin and red corpuscles are more likely to be equally diminished, and the blood-plasma also becomes less albuminous, but it is only in the extreme cases that this holds, and it is necessary to inquire very carefully into the patient's history. No amount of iron will take the place of sleep or fresh air in treatment.

(7) *Kidney Disease*—Chronic nephritis always causes anaemia in young people, but the blood-plasma contains a smaller amount of solids. The examination of the urine usually clears up the diagnosis.

(8) *Anæmia from Chronic concealed Haemorrhage*—This may sometimes be from piles, of which the patient, from ignorance or modesty, does not tell her doctor, but more often from ulcer of the stomach or duodenum. These ulcers may give rise to no symptoms, or to none that are not usual in chlorosis, and the blood in the stools is so much altered that the lay public cannot recognise it. The blood examination gives little or no help here, and there will be need of a very careful review of the whole case, and repeated examination of the stools.

(9) *Early Pregnancy*—This must always be borne in mind as a possible cause of amenorrhœa and anaemia in young girls.

**TREATMENT**—(1) *Prophylactic*—In families where chlorosis has occurred in the elder sisters, it is worth while to be specially careful of the health of the younger sisters during the years when they are likely to be attacked. From what has been said with regard to the etiology of the disease it will be evident what the necessary measures are—a healthy life, with sufficient food, air, and exercise. It is quite useless to give iron before the disease develops, as it does not prevent the onset of chlorosis, and, if the system has become habituated to its use, it may fail entirely to assist blood formation when it is really needed.

(2) *General*—Sunlight and fresh air are of prime importance, and too much attention cannot be bestowed on them. Rest is also an all-important factor. All severe cases should be sent to bed, and so should all cases of medium intensity whose circumstances will allow of it. This removes the strain on the enfeebled and badly-nourished heart, and the symptoms of dyspnoea, faintness, headache, and neuralgia are relieved almost immediately. The length of time that the patient is to remain in bed depends on the severity of the case and the success of treatment, generally three weeks is a sufficiently long time, but it should be some

weeks longer before she is allowed to resume her ordinary duties. In slight cases, where it is not necessary to make the patient go to bed, she should rest as much as possible, avoid fatigue and excitement of all kinds, and keep early hours.

The diet should be regulated to suit each case, of course, but regularity of meal times should be insisted on, and the want of appetite and disinclination for food can often best be dealt with by ordering relatively small meals at shorter intervals than usual—say every three hours. The distaste for meat which almost all chlorotics evince must be overcome, gently but firmly, and an ordered quantity of it, small at first, but increasing, must be taken. The previous diet has often consisted of bulky but unnutritious foods, and these should be replaced by proteids, whose concentrated nourishment can be more rapidly utilised. When the patient is thin, milk with an equal quantity of cream added to it may be given in addition to the meat, and where patients cannot take solid food milk will be given largely, but to fat patients, and to those who have a good appetite for solid food, a large quantity of milk should not be given. Alcohol, which favours fat formation, may be given to thin patients. The popular superstition that claret and other red wines “make blood” is of course absolutely groundless. Cold bathing, or anything else which withdraws heat or causes shock to the heart, should be avoided. The bowels must be carefully regulated, as iron has a tendency to produce constipation, though this is often overrated.

(3) *Special*—Iron cures chlorosis in the great majority of cases. It does not do so by replacing lost or diminished iron in the blood. The iron-containing proteids of the food are quite capable of doing this, but rest and good food alone will not cure chlorosis. What is necessary is a very active stimulation of the bone-marrow, and the salts of iron circulating in the blood are the best stimulant to the marrow. The form in which iron is to be taken is to a certain extent a matter of indifference, and depends upon the digestive powers of the patient, our ability to give a sufficient amount of iron without giving too bulky a dose of the preparation, the experience of the physician with similar cases, and only secondarily on the special properties of individual iron preparations. All of them, organic and inorganic, are transformed in the stomach into ferric chloride. It was supposed that the inorganic salts of iron were not absorbed, and as a result there were put upon the market numerous preparations of iron-containing nucleo-albumins and proteids, under the names of hæmoferrum, hæmol, hæmatogen, hæmoglobin, carniferin, ferratin, etc. These contain iron in organic combination, and were supposed to be more easily assimilable than the inorganic salts. They are all, however, too much like food-iron, do not stimulate the

marrow with the same rapidity or vigour as the inorganic salts, and many of them have the further disadvantage that they contain so small an amount of iron that enormous doses would have to be taken to make up the necessary minimum daily dose, which is from 0.1 to 0.2 grammes of metallic iron. Of the inorganic salts, which we now know can be absorbed, the one which is most used is the carbonate, in the form of Bland's pill or capsules, or as the saccharated carbonate. It is not astringent, and is generally well taken. The proto-sulphate and the ferric salts are astringent, and cannot usually be taken by people with irritable stomachs, but where the digestion is good they are often very useful. Best of all, perhaps, is reduced iron, provided it does not contain sulphur as an impurity; when it is dissolved in the gastric juice hydrogen is evolved, and if sulphur be present, unpleasant eructations of sulphuretted hydrogen result. Its small dose is an advantage. The scale preparations are sometimes useful with dyspeptics, as they are easily taken, but they contain a relatively small amount of iron.

The dosage should be carefully regulated. Bland's pill may be taken as a standard, and of these six a day, two after each meal, should at first be given. After three or four days or a week the number may be doubled, if there is no increase of gastric disturbance, and after another week the number may again be increased. It is rarely necessary to give more than from 20 to 24 pills a day, but the maximum, when once reached, should be persevered with for some weeks, and the dose then gradually decreased for a fortnight before leaving off altogether. The length of time during which the iron is to be taken varies in different cases, and should be determined by the effect on the blood. As long as the hæmoglobin percentage improves the iron should be continued, but if it becomes stationary it is best to stop the iron, let the patient go without any for from two to four weeks, and then begin again in the same way. It is generally a mistake to let a patient go on taking iron indefinitely, as the system becomes habituated to its use, and if a relapse occurs treatment is not so effectual. Iron must always be given after food; no matter what form is taken, it must be taken regularly and without interruptions, and as the dyspepsia of chlorosis is usually due to their anæmia, iron should be given even though the patient complains of slight dyspeptic symptoms. It may sometimes be necessary to give a bitter tonic, or some such remedy as bismuth, rhubarb, and soda before food for a few days, while the iron is being given, and it is often wise to begin treatment with a purge.

In cases where the iron is apparently doing no good it is well to see that the preparation which is being taken is really active, to see, for instance, that the pills are soluble, or to change

the preparation. Chalybeate waters are sometimes useful in such cases, or arsenic may be tried along with or instead of the iron. It is most likely to be useful in cases where the number of blood corpuscles is greatly reduced, and it must be given at first in small doses and gradually increased. Blood-letting has been used as a means of treatment, and might be tried in those very chronic cases where drugs are not very successful. Hæmorrhage is a powerful stimulant to the marrow, and may give the necessary fillip to blood regeneration which can then be maintained by iron. About four ounces of blood might be drawn.

Treatment should in all cases be continued until the normal hæmoglobin percentage is reached, and it must be remembered that patients often recover a healthy appearance long before this result is attained.

**Chloros.**—A preparation resembling chloride of lime (bleaching-powder), used as a disinfectant; it contains 10 per cent of available chlorine.

**Chlorozone.**—A yellowish liquid acting as a bleaching agent and disinfectant; it is formed by passing nascent hydrogen (mixed with air) into caustic soda.

**Chloryl.**—A mixture of methyl chloride and ethyl chloride; a local anæsthetic.

**Choanæ.**—The posterior openings of the nares. See *NOSE, EXAMINATION*, etc.

**Chocolate.**—Ground cocoa, with sugar, etc., added, and with some of the fat removed. Like cheese, it contains much nutriment in small compass. See *DIET (Fruit, Nuts)*.

**Choke Damp.** See *TOXICOLOGY (Gaseous Poisons, Carbon Dioxide)*.

**Choked Disc.**—Projection of the optic papilla above the level of the retina (as seen by the ophthalmoscope), with extension and blurring of its border, and increased vascularity, due to increased intracranial pressure (cerebral tumour, nephritis); marked "papillitis." See *RETINA AND OPTIC NERVE (Optic Nerve, Inflammation)*.

**Choking.** See *ASPHYXIA (Causes)*; *MEDICINE, FORENSIC (Death from Asphyxia, Suffocation)*; *ESOPHAGUS (Foreign Bodies)*.

**Cholæmia.**—The presence, in excessive amount, of bile in the blood; jaundice. See *JAUNDICE (Pathology)*.

**Cholagogues.**—A class of medicinal substances, including podophyllum, euonymin, iridin, sodium salicylate, mercury (calomel), colchicum, and most of the cathartic purgatives; they act either directly by increasing the secretion of bile, or indirectly by stimulating the

action of the upper part of the small intestine, and so carrying the bile down below the level of the bowel where reabsorption occurs, such medicines ought to be followed by a saline. See PHARMACOLOGY, and under names of various drugs

**Cholangiostomy.**—Formation of a gall-bladder fistula

**Cholangiotomy.**—Incision of a bile duct (intrahepatic) for the removal of gall-stones

**Cholangitis.**—Inflammation of the bile ducts, *e.g.* due to gall-stone in the common duct. See GALL-BLADDER AND BILE DUCTS, DISEASES (*Cholelithiasis, Cholangitis*), LIVER, DISEASES (*Hypertrophic Biliary Cirrhosis*), LIVER, DISEASES (*Hepatic Tuberculosis*), LIVER (*Tropical Abscess, Diagnosis*), PANCREAS, DISEASES (*Malignant Disease*)

**Chole.**—In compound words *chole-* (from *χολή*, bile) signifies *relating to the bile*. In addition to the words specially dealt with below, the following may be named *cholechyus* (discharge of bile), *cholecyst* (the gall-bladder), *cholecystitis* (inflammation of the gall-bladder), *choledochitis* (inflammation of the common bile duct or ductus communis choledochus), *choledochostomy* (the production of a fistula of the common bile duct), *choleic acid* (taurocholic acid), etc.

**Cholecystectomy.**—The removal of the gall-bladder, in whole or in part. See GALL-BLADDER AND BILE DUCTS, DISEASES (*Tumours of Gall-Bladder, Treatment*)

**Cholecystendysis.**—The excision of a gall-stone from the gall-bladder, followed by closure (by sutures) of the opening in the gall-bladder, and by the anchoring of the bladder to the abdominal incision, which is also closed

**Cholecystenterostomy.**—The establishment of an artificial communication between the gall-bladder and the intestine (*e.g.* the duodenum) in the treatment of gall-stones in the common duct. See GALL-BLADDER AND BILE DUCTS, DISEASES (*Cholelithiasis, Treatment, Tumours of the Gall-Bladder, Treatment*)

**Cholecystitis.**—Inflammation of the gall-bladder. See GALL-BLADDER AND BILE DUCTS, DISEASES (*Cholelithiasis*)

**Cholecystostomy.**—The making of an opening into the gall-bladder and the bringing of it into an opening in the abdominal wall (or intestine) and fixing it there

**Cholecystotomy.**—An operation in which the abdomen is opened and the gall-bladder incised for the removal of gall-stones or some other purpose, the incision may be closed again, or the bladder may be fixed to the abdomi-

nal wound, establishing a fistula. See GALL-BLADDER AND BILE DUCTS, DISEASES (*Cholelithiasis*)

**Choledochotomy.**—The opening of the abdomen and the incision of the common bile duct for gall-stones in that duct, then the opening may be closed by sutures, or it may be brought to the abdominal incision and a fistula established (*choledochostomy*), or it may be made to open into the intestine (*choledochenterostomy*). See GALL-BLADDER AND BILE DUCTS, DISEASES (*Cholelithiasis, Treatment*)

**Cholelithiasis.**—Gall-stones, the diseased condition caused by their presence in the gall-bladder, in the cystic duct, or in the common duct. See GALL-BLADDER AND BILE DUCTS, DISEASES (*Cholelithiasis*)

**Cholera, Asiatic.** See CHOLERA, EPIDEMIC

**Cholera, Epidemic.**

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See also AIR, EXAMINATION (*Ground Air*), EPIDEMIOLOGY, FACES (*in certain Diseases*), GASTRO-INTESTINAL DISORDERS, IMMUNITY (*Cholera*), METEOROLOGY (*Seasonal Prevalence*), MICRO ORGANISMS, WATER (*Diseases produced by*)

SYNONYMS—*Asiatic cholera*, *Fr cholera*, *Ger Breichruhr*, *Gallensucht*, *It colera asiatico*. The Indian names are *monshi*, *mondesian*, *vu-chika* or *buuche*, *hazza*, *aitangui*, *uaba*, etc.

DERIVATION—The word cholera was applied by the Greek and Roman physicians to the disease known as summer cholera or cholera nostras. Its etymology is uncertain. Celsus supposed it to be derived from *χολή*, bile, and *ῥέω*, to flow, others, with Alexander of Tralles, derive it from *χολάδες*, the intestine, and *ῥέω*, to flow, while some recent authorities incline to the view that it comes from *χολέρα*, the gutter of a roof

BACTERIOLOGY—In 1884 Koch discovered the comma bacillus or vibrio, which is now generally recognised as the *ens morbi* of cholera. Pure cultures of the microbe are undoubtedly capable of giving rise to the disease in man, and if the evidence derived from the experimental production of the cholera in the lower animals is more ambiguous, this arises from the fact that cholera is essentially a human disease, which does not give rise to the same train of symptoms in the lower animals that it does in man. So much unanimity now obtains respect-

ing the pathogenic character of the vibrio, that it is unnecessary to recount the accidents and experiments which establish beyond doubt that all the symptoms and lesions of cholera can be caused by it in man, or to discuss the value of the evidence afforded by experiments on the lower animals.

The vibrio is found in the stools of cholera patients, and after death in the contents and tissues of the intestinal canal. It has occasionally been detected in the vomited matters, but only in small numbers, and its presence in the vomit is doubtless to be accounted for by the contents of the intestine finding their way into the stomach. It has also been met with in a few instances in the bile ducts and gall-bladder. It is never present in the blood, liver, spleen, kidneys, or mesenteric glands. The real seat of the microbe is the lower part of the small intestine. Abcl and Clausen made the important observation that vibrios are often present in the stools of healthy persons who are in daily intercourse with cholera patients. They have also been found in the stools of convalescents up to fifty days after recovery.

When one of the small mucous flakes from a cholera stool is spread out on a cover-glass, dried, heated, and stained, the bacillus is readily to be seen if the specimen happens to be a pretty pure cultivation, otherwise the presence of other organisms makes its detection difficult. In many cases its presence or absence can only be certainly determined by placing a mucous flake in peptone broth, and incubating for twenty-four hours, the vibrios in sufficiently pure culture for microscopic examination are then to be found on the surface of the fluid. The various media and processes of cultivation, and the distinctive characters of cholera colonies, will be found described in any text-book on bacteriology.

The cholera bacilli are actively motile, flagellated, curved rods, about half the size of the tubercle bacillus. Their average length is about  $1.5 \mu$ , their thickness, from one-sixth to one-third their length. The young forms show only a slight curve, the older ones a more decided bend, while some present the form of a half-circle. They occur isolated or attached to one another in the form of the letter S, or in longer screw-like forms. They are stained with warm solutions of methylene blue, or with methyl-violet, or fuchsine.

They do not thrive in acid media. They are aerobic, but are nevertheless capable of growing to some extent when oxygen is altogether excluded. The cultivations made with a deficient supply of oxygen are more virulent, but less resistant to the action of the gastric juice and external influences than those grown with a more abundant supply of oxygen. It is the circumstance that they are capable of growing when only a slight amount of oxygen is present

that explains their rapid multiplication in the intestinal canal. This may also explain their exalted toxic power as parasites, a toxicity which they lose when grown for a time as saprophytes with a free supply of oxygen.

The cholera bacillus does not grow below  $16^{\circ}$ , it thrives between  $22^{\circ}$  and  $25^{\circ}$ , its optimum lies between  $30^{\circ}$  and  $40^{\circ}$  C. It is killed by exposure for half an hour to a temperature of  $60^{\circ}$ . Although the bacillus does not grow below  $16^{\circ}$ , Koch found that it is not killed by being subjected for an hour to a temperature of  $-10^{\circ}$  C. It is very susceptible to drying. Koch says that when spread out on a cover-glass and exposed to the air, the bacillus is killed after two or three hours, but according to Kanthack the vibrios have been found to retain their vitality for 120 days when dried on glass. When exposed to sun and air they do not live long on a cover-glass, but if the air is humid, and the preparation is not exposed to sunlight, they are not so easily destroyed.

Commas are easily destroyed by the growth of saprophytes, but experiments bearing on this point give widely different results. The nature of the medium, its reaction, and the class of organisms it contains all no doubt count for much. Koch found that when added to sewage the vibrios could not be demonstrated after twenty-four hours, on the other hand, they have been observed to retain their vitality for months in sewage-polluted water. Koch did not find them to survive longer than six or seven days in the water of the Berlin Canal. Orgel found that they could live for nearly twelve months in ordinary Elbe water. They grow at first very luxuriantly in moist soil, but after a few days they generally succumb to saprophytes. They retain their vitality for a long time on moist linen, on which they are often found in a state of pure culture. The vibrio is not very fastidious as regards food. It grows on agar-agar to which meat infusion and peptone have been added, in slightly alkaline nutrient gelatine, on solidified blood serum, on potatoes, milk, and, to some slight extent, even in sterilised water. This shows its adaptation to saprophytic growth.

So far we have been dealing with the characters and habits of the microbe as observed in experiments. Little is known of its life-history outside the human body, but what little we know shows that it can retain its vitality longer than cultivation experiments would lead us to anticipate. The microbe under natural conditions is a hardier plant than many suppose. In the sewage-polluted water of Marseilles harbour it has been found to survive for eighty-one days. There is reason to believe that it may exist not for the few days which experiments indicate for its limit, but for months, perhaps for years, in the soil (see article on "Epidemiology").

But while the cholera vibrio is capable of retaining its vitality, it loses much of its virulence when grown for a time as a saprophyte, and the more vigorous its saprophytic growth, the less its virulence. From Hankin's observations this loss of virulence is observed even in India, where the conditions might be supposed to be peculiarly favourable for it retaining its properties. Not only does it lose in virulence when grown continuously outside the body, but it also undergoes marked morphological changes.

It is admitted that the vibrios met with in cholera stools present considerable differences, which tend to perpetuate themselves through successive generations. Some have one flagellum, others more. Less definite distinctions in size and form are also observed. Differences, too, exist in the appearances presented by the colonies they form on gelatine plates and in their power of liquefying gelatine, and all this has given rise to discussions as to the unity or multiplicity of the germ. The marked morphological and biological changes in vibrios, derived from a common stock, which are observed to occur under cultivation, prove the variability of the organism, but the essential unity of all the varieties is attested by their common pathogenic properties, and by the immunity which the vaccine of one variety confers from attacks of all the others.

When the cholera vibrio is cultivated in the peritoneal cavity of the guinea-pig its virulence is increased, and it is by passing it in this way through a succession of guinea-pigs that Haffkine obtains his prophylactic virus. A culture from the peritoneum furnishes a pure subculture on agar, which is thoroughly shaken up with broth. This constitutes the vaccinating fluid, which may be used as a living vaccine, or the bacillus may be killed before being used.

Very great interest and importance attaches to the investigations of Metschnikoff bearing on the influence of non-pathogenic organisms in promoting or hindering the development of the cholera vibrio on external media, and in determining infection in animals. He found that some species of sarcinae, torulae, and a variety of the colon bacillus promoted the infection of suckling rabbits. The associated organisms rapidly disappeared, but none the less they subserved to the development of the cholera virus in these animals. When we remember the effect of moulds in promoting the growth of the bacillus of yellow fever, and of streptococci in intensifying the virulence of the diphtheria bacillus, we feel that it is in this association of non-pathogenic organism with the cholera bacillus that we are likely to find an explanation of some obscure points in the etiology and epidemiology of the disease.

The manner in which the comma bacillus is influenced by temperature, moisture, rainfall,

and drought throws considerable light on some of the epidemic features of cholera.

(a) The relation of the vibrio to temperature explains why in temperate climates cholera epidemics are mostly restricted to summer and autumn. It is in these seasons only that the temperature permits its saprophytic growth.

(b) The effect of drying on the vitality of the parasite accounts for cholera dying out during the long dry season in the Punjab and Central Provinces, and its reappearance after the rain begins to fall, when the earth and atmosphere become moist and humid. In such regions the cholera season is regulated not by the temperature as in Europe, but by the rains.

(c) The inability of the vibrio to live for any length of time in fluids deficient in nutritive material, as well as the effect of excessive moisture of the soil in reducing the oxygen at the disposal of the organism, explain the subsidence of the disease in endemic areas, when the heavy rains submerge large tracts of land, and displace the air from the soil that remains uncovered.

**ETIOLOGY.**—For a widespread epidemic of cholera the following conditions are necessary—(1) The presence of the microbe, (2) a suitable medium and temperature for its growth outside the body, (3) means of transport from place to place, (4) a vehicle by which it can be diffused in a particular locality, (5) a susceptibility for infection on the part of a community.

1 *Sources of the Virus.*—The primary source of the virus, outside the regions in which cholera is endemic, is in every instance the intestinal discharges of a person suffering from, or one who has recently suffered from the disease, or of a person who without suffering from the disease harbours the microbe, from having associated with a cholera patient.

It will seldom happen, however, that the microbe grown in the intestinal canal of a cholera patient will find its way directly into that of a healthy person, for cholera discharges will only be swallowed accidentally, or as the result of a scientific experiment. It is the descendants of this microbe grown in some external medium—water, milk, soil, linen—which in most cases causes infection.

2 *Breeding-places.*—The virus having been introduced into a locality must find some medium outside man in which it can grow. Such media are (a) a soil polluted with organic matters, especially excreta, under soils we include accumulations of animal and vegetable refuse, cesspools, etc., (b) sewage-polluted water, (c) milk and other articles of food.

Having found a suitable medium, the bacillus requires a certain temperature for its growth. Cholera was introduced simultaneously into New York and New Orleans in December 1846, and broke out in both cities, but in New York the outbreak rapidly subsided, whereas in New Orleans it spread during the winter. This

difference will be easily understood when we remember that the winter temperature of New York is under freezing-point, while that of New Orleans is about 60° F. How favourable soever other circumstances may be, an extensive epidemic of cholera cannot occur in winter in higher latitudes, except under very exceptional conditions.

3 *Means of Transport*—The view that the cholera virus can be transported for long distances by the air is quite untenable. Byden maintained that atmospheric moisture is the carrier of the virus, and that its distribution over India extends as far as the moisture-laden monsoon winds carry it. The rains supplied by the monsoon act in a different way, they bring the soil into a condition fitted for the spread of the pestilence. The germs of the disease are always being carried from Bengal to the north-west, but they do not develop epidemic outbreaks until the condition of the soil favours the growth of the microbe.

The principal means by which the virus is transported from place to place, so as to overrun vast regions, are (a) *human intercourse*, that is, by persons who have, or have had, the disease, or who harbour the vibrio, and things contaminated by cholera discharges. That this is the principal means of its propagation is proved by the constancy with which cholera has followed caravan routes and lines of communication by river, road, rail, or ship, and by its rapidity of spread increasing as intercourse between distant countries becomes more rapid. In its early invasions of Europe, cholera followed the routes of caravan traffic. Cabul has always received the infection from India, and has been the centre from which it has advanced westwards. One route from Cabul passed through Central Asia, by Balkh, Bokhara, and Khiva to Orenburg, another through Persia by Herat, Mesched, Astrabad, Teheran, Reshed, Baku, and Astrakhan. An alternative route from Teheran led through Tabreez, Tiflis, Erzeroum, and Trebizond to the Black Sea. In the same way cholera invariably breaks out in an island at ports in communication with an infected place. In Russia in the olden time cholera followed the rivers, and on its first outbreak in England in 1832 it followed, as Hirsch points out, "the commercial highways chiefly, and the coast routes and rivers, while the mountainous parts of the country were little visited by it, and the Scottish Highlands not at all."

When we remember that cholera has been five times epidemic in Mauritius, and that on each occasion it has broken out shortly after the arrival of vessels from India with the disease on board, that it broke out at Quebec in 1832 four days after the arrival of the *Carrick*, on board which cholera had prevailed during the voyage (and the Western Hemisphere had never up to that day been visited by the disease), that it appeared at New York in 1848, which was then free from the infection, on the arrival of a vessel

which had lost seven passengers from cholera; that it appeared at New Orleans in the same year, three days after the arrival of the ship *Swanton*, thirteen of whose passengers had died of cholera during the passage, and when we see it breaking out along the routes of pilgrims, tracking the march of armies, following the lines of emigration, we will be driven to the conclusion that human intercourse is the most important of all the means by which cholera is transported from place to place, from one country to another, across deserts and oceans.

(b) *Rivers*—A stream polluted by cholera discharges may carry the virus for very considerable distances to towns situated on its banks. In the last outbreak in Germany the frontier river Premsa, a tributary of the Vistula, had become polluted by the cholera virus. Shortly afterward, cases began to appear among the river raftsmen and bargemen on the Vistula. The cholera bacillus was found in the water of the river above Dantzig, where cases now began to appear. By the middle of June at least six German towns and villages on the banks of the river had become infected (see *Local Government Report*, 1897-98).

(c) *Ships*, which are moving centres of infection, transport the virus across oceans. This usually happens through the agency of infected persons, but it may also take place by means of infected goods, ballast, or bilge water.

4 The diffusion of cholera in a given locality is a question distinct from that of its transport.

(a) The vehicle by which it is most frequently conveyed into the system is *drinking water*. When the virus finds its way into the general water-supply of a city, as was the case in Hamburg in 1892, the disease becomes rapidly and widely diffused. When wells become polluted we have local outbreaks, as happened in the well-known Broad Street explosion in London in 1854, in which the disease was practically confined to those who made use of a contaminated well, and ceased from the day on which the well was closed.

The important part played by water in the spread of cholera is seen in the decrease of the disease in towns in which it was formerly prevalent on the introduction of a pure water-supply. The average mortality of the European troops at Fort-Willem, Calcutta, was 20 per 1000 from 1826 to 1863. From the latter date, when the fort was for the first time supplied with pure water, to the present time it has averaged 1 per 1000.

Lahore had an average cholera death-rate of 1.07 per 1000 for the fifteen years 1848-61. In the period 1882-87—that is, after the introduction of water into the city from the Ravi River—it fell to 0.07 per 1000. That the reduction in the cholera death-rate has really been due to the improved water-supply is proved by the fact that in the Lahore district (excluding the city) the cholera mortality, which was 0.34 in

the former series of years, rose in the latter period to 0.43 per 1000

(b) *Milk and other articles of food* are not only media for the growth of the virus, but vehicles for its diffusion. Insects doubtless play an important part in spreading the disease by settling on articles of food after having been in contact with substances containing the virus. The house-fly in particular is an active agent in disseminating the germs of the disease in this way, and now that it has been shown that the vibrio is capable of living for at least fourteen days in the fly, wider limits must be assigned to its pernicious activity than was formerly conceded to it

(c) *Air as a Vehicle of Infection*—Hirsch, writing in 1883, says, "The facts do not permit us to ignore that the poison *must* be taken up and suspended in the air, so as to enter the human organism with the breath." This was how the facts looked to so eminent an epidemiologist a few years ago. The facts remain the same, but the way in which they are looked at and interpreted has so completely changed, that to many it seems now incredible that the virus ever enters the system by means of the breathing air. It is held that micro-organisms can only be detached from perfectly dry surfaces, so as to be carried about with dust in the atmosphere, and as the cholera vibrio is supposed to be unable to bear this amount of drying, infection through the atmosphere is regarded as impossible. The premises are doubtful, and the conclusion contrary to well-observed facts. That cholera is not usually diffused by means of the air is evident enough, but air is nevertheless one of the vehicles by which the virus enters the economy. It is not to be supposed that infection takes place through the lungs. The vibrio lodged in the upper air-passages may live or even multiply in the alkaline mucosities of the parts, and then be conveyed into the stomach along with food.

As space forbids us entering into detail, we shall content ourselves by briefly referring to two categories of facts illustrating the occasional aerial convection of the cholera virus.

To the first category belong particular instances in which the infection has been carried from the sick-room, and those in which simple proximity to a source of infection has given rise to the disease. Copland records an instance in which all the circumstances seemed to demonstrate that he carried the infection on his clothes for a distance of about a mile and a half and communicated the disease to two of his relatives (*Dictionary*, article "Epidemics"). Similar instances are by no means rare. Simple proximity to a source of infection may also give rise to infection. Cholera broke out on the steamship *England* from Liverpool. There had been 150 cases and 48 deaths on board when she bore up for Halifax. A pilot hailed the vessel, but

having learned that there was a fatal disease on board, he laid his boat close alongside, sent up his papers to the captain in a basket that had been lowered from the ship, and brought the vessel up to the quarantine station *without having boarded her*, and then rowed ashore with his two comrades. Two days after having come thus remotely into contact with the *England* he was taken ill of cholera, and three days afterwards cholera broke out in his family. Almost at the same time one of his two companions sickened and gave the disease to three of his children. The Western Hemisphere had then been three years free from cholera (Hirsch). An instance of approach to a dead body being followed by cholera is given by Clemow in the *Transactions of the Epidemiological Society* for 1893-94.

To the second category belong the sudden explosions of a number of cases of cholera on board vessels in which the disease had before been occurring in a sporadic way, after a storm during which the ports and other means of ventilation have had to be closed. These scarcely admit of any other explanation. Instances of this kind are given in detail by Smart.

The *Britannia* ship of war, for example, in 1854 was infected while lying at Buljick, where cholera existed. Up to the 10th of July three deaths had occurred. She put to sea on the 12th, and the disease seemed to subside at once. On the evening of the 13th a gale necessitated the closing of the ports on the sleeping-deck. About 10 A.M. on the 14th "a great and sudden outburst of collapsed cases occurred." There is nothing in the circumstances in such instances (and they are numerous) to incriminate water or food, but everything points to the virus diffused through the air of crowded and unventilated holds causing these sudden explosions.

5 Individual susceptibility counts for much in the matter of infection. It is only a small proportion of a community that is attacked during an epidemic. Excesses of all kinds, especially alcoholic excesses, causing gastro-intestinal catarrh, predispose to cholera. A community acquires an immunity lasting for three or four years after passing through a severe epidemic of cholera.

A few circumstances relating to the etiology of cholera require to be mentioned—

(a) *Topographical Relations*.—Cholera shows a special predilection for the low-lying parts of a town. Farr laid it down as a law "that the proportion of deaths from cholera is inversely as the elevation of the ground," and this law holds good excepting in instances in which the incidence of the disease is determined by the contamination of a water-supply. As an endemic disease, cholera is limited to altitudes not exceeding 1500 feet. As an epidemic malady it has broken out at elevations of 6000 feet (Kissoulh, 1845).

(b) *Racial Relations*.—The following proportions per cent were attacked at Guadeloupe in 1865:—

Chinese	2.7
Whites	4.31
Hindooes	3.26
Mulattoes	6.31
Negroes	9.44

The comparative immunity of the Chinese has also been noticed in Mauritius, and has been ascribed to their opium-eating habits, but it is to be remembered at the same time that nowhere has cholera raged more destructively than in the Chinese Empire.

(c) *Personal Relations*.—Sex has no influence on the liability to cholera. Its incidence on different ages varies in different outbreaks. In the Hamburg outbreak of 1892 the ages fifteen to twenty-five gave the smallest ratio of attacks and also of deaths to the number attacked. Children under two years of age suffered considerably, as did also old persons. The poor, as a rule, suffer more than the rich, and a special liability attaches to the inmates of Lunatic Asylums. As respects occupations, little can be said, except that washerwomen employed in washing cholera linen contract the disease out of all proportion to its incidence on the community generally.

*Exempted Places*.—Some places seem to be proof against cholera. It has never appeared in Cheltenham, Seclau, or Würzburg. In other places, such as Versailles, Lyons, and Martinique, the disease has never assumed epidemic proportions.

#### MORBID ANATOMY

When death occurs at the height of the disease, rigor mortis is well marked, the features are pinched, the face, extremities, and body generally are more or less cyanotic.

The cerebral sinuses and the veins of the meninges contain dark blood.

The pleurae are dry, and frequently present numerous ecchymoses. The lungs are dry and collapsed, and are much below the normal weight. The larger branches of the pulmonary arteries contain blood, the smaller arteries, capillaries, and pulmonary veins are empty.

The pericardium does not contain a trace of serum. The visceral layer is frequently studded with small ecchymoses. The right side of the heart and the veins emptying into it, as well as the jugular veins, the portal vein, and the larger hepatic veins, are engorged. The left side of the heart is empty and contracted.

The stomach is empty, the lining membrane may be pale and sodden or congested. Occasionally it presents ecchymotic points. The duodenum and jejunum are frequently hypersemic continuously or in patches.

The large intestine is contracted. The peri-

toneum covering the small intestine has often a rosy appearance and is dry and sticky. The ileum usually contains more or less rice-water fluid, and is throughout, especially in its lower part, congested and oedematous, but these appearances are most marked in its lower half. Occasionally the mucous membrane is found pale, instead of congested; at other times ecchymosed and suffused. The solitary glands and Peyer's patches are prominent, the latter often surrounded by a red zone of congestion. On microscopic examination the mucous membrane in many parts is found denuded of epithelium. At other places the epithelium is detached from the subjacent tissues by serous exudation. Commas are found in the tubular glands, and between the epithelium and the basement membrane, on the surface of, and sometimes within the villi, and occasionally also in the deeper tissues of the mucosa. The mesenteric glands are swollen and softened.

The liver is often somewhat increased in volume, dark, congested, but of normal consistence. The gall-bladder is full of bile of varying viscosity and colour. The spleen is small, dry, and anemic.

The kidneys, when death occurs early, are often augmented in volume, and in this case the medullary and cortical substances show punctuated, patchy, or striped areas of venous congestion, occasionally ecchymotic points or patches. The vessels of the glomeruli are congested. In other cases the congestion is less marked, and the kidneys may even be pale. The epithelium of the tubules is swollen and cloudy, blocking up the lumen of the tubes. The bladder is empty and contracted.

When death has occurred during the stage of reaction, the appearances are altogether different from those just described, and the lesions vary considerably according to the symptoms developed during the reaction.

The cerebral membranes are injected, and there is often effusion into the meshes of the pia mater and into the lateral ventricles. The lungs are congested, and the pneumonic or other inflammatory lesions may be present. The mucous membrane of the small intestine may be softened, ulcerated, or covered with patches of diphtheritic exudation, and this may extend to the large intestine. The liver and spleen are usually somewhat enlarged and congested, the kidneys enlarged, vascular, of a dark red colour, with fatty and granular casts in the canaliculi. The urine in the bladder is generally albuminous.

#### SYMPTOMS

The cardinal symptoms of cholera are diarrhoea, vomiting, muscular cramps, paresis of the heart, algidity, suppression of urine, followed by death or reaction.

The attack may begin with a diarrhoea in-



distinguishable from an ordinary diarrhoea except by bacteriological examination of the discharges. This premonitory diarrhoea usually lasts from half a day to two days before the choleraic symptoms declare themselves, and when promptly treated, the disease is frequently arrested at this stage. This premonitory stage is often enough absent.

The actual attack begins with frequent, copious, watery evacuations, at first coloured with bile, but soon becoming pale, having an alkaline reaction. Along with this there is urgent thirst, great discomfort in the bowels, an indescribable feeling at once of intolerable distension of the abdomen and sinking that cannot be understood by those who have not experienced it, nor forgotten by those who have. There is no tenesmus.

The stools become more frequent and copious. They flow in streams. No sooner has the patient evacuated, as he thinks, the whole contents of the bowel and hopes to obtain a little respite, than he is again disturbed, and again passes an enormous quantity of an odourless, colourless, rice-water liquid, which on rest deposits fine, flaky particles.

After a time vomiting sets in, and so much the sooner, the more freely the patient has gratified his craving for water. Large quantities of a pale liquid are ejected forcibly, but without effort, from the mouth. It seems as if it were automatically pumped out. The patient is now extremely restless, tosses off the bed-clothes, and although his skin feels cool or cold, he complains of heat. The pulse is rapid and weak, the heart's action feeble. The extremities become cold and blue, the fingers shrivelled and livid, the temperature in the axilla may fall to 94° or 93° F., sometimes lower, that of the rectum is several degrees above the normal, and it may be well to remark that the rectal temperature is often higher than normal before the algid state has declared itself. It happens, however, in many cases that the rectal temperature follows closely that of the axilla. The voice becomes weak and hoarse—the so-called *vox cholerae*, the eyes are sunken, the eyelids half closed, the face pinched, the breath feels as if it had passed over ice, the tongue gives, as Dr Watson says, the sensation as if one were touching a frog's belly, the urine is scanty or suppressed. This is the algid stage of cholera.

When this state has begun to develop, or even before, painful muscular cramps of the calves, arms, and sometimes of the abdominal muscles, set in, which add greatly to the sufferings of the patient. They are tonic spasms, lasting for two or three minutes.

When the algid state is established, the motions usually become scantier and less frequent, and are passed in bed. Or they may entirely cease, being retained from a paralysed state of the bowel—a symptom of evil omen.

Retching, alternating with hiccup, takes the place of the vomiting.

This state may last for a few hours only, or persist for one or even two days—perhaps eight to fifteen hours is the average—and terminates in reaction or in death.

When the disease is tending to a fatal issue, the patient sinks into an apathetic state, heedless of what is passing around him, but at the same time conscious. The heart's action grows weaker, the pulse imperceptible at the wrist, scarcely to be felt even in the carotid, the skin becomes covered with a clammy sweat, and the patient lies in a state of collapse.

Before the algid stage has set in, or after it has been established, things may take a favourable turn by the setting in of reaction. The pulse becomes fuller, slower, the impulse of the heart stronger, the breathing deeper, easier, and less hurried, the rectal temperature falls, and it is only after this that the surface regains its warmth. The cramps disappear, the retching and purging subside, and the patient falls into a tranquil sleep, from which he awakes refreshed. It is often not until after thirty-six to forty-eight hours that urine is passed. The first urine is of low specific gravity, containing more or less albumin, sometimes blood. In favourable cases the recovery is rapid.

But reaction does not necessarily imply that all danger is over, for in many cases it introduces a new series of troubles. The reaction may be imperfect, the discharges recur, thirst returns, and the patient, when we were beginning to hope for recovery, falls back into the algid stage.

In other cases he lapses into a typhoid condition marked by great debility, more or less stupor, restlessness, especially at night. There is complete anorexia, occasional vomiting, diarrhoea or constipation, or both alternately, with metemesis, scanty albuminous urine, containing fibrinous casts. After this condition has lasted from four to seven days, the symptoms may gradually improve, in this case the appetite returns, the urine becomes more abundant, the albumin diminishes, and the head symptoms pass off.

In other cases the symptoms become aggravated, and the patient falls into a comatose state. It has to be mentioned that typhoid symptoms terminating in death from coma occasionally occur, although the urine is abundant and free from albumin. This typhoid condition is all the more likely to supervene the longer the algid state is prolonged. Those who have been addicted to drink seldom recover without exhibiting typhoid symptoms, which in their case are even more dangerous than in others. Gangrene of the penis, scrotum, nose, and of the mucous membranes of the mouth are the rarer sequelae of cholera. More frequently the parotid glands become swollen and inflamed, and sometimes suppurate.

The duration of the disease in fatal cases varies greatly. A few die from four to eight hours after the commencement of the attack, more from twelve to forty-eight hours. The algid state seldom lasts beyond the second day. Those who survive this, die in the typhoid stage from the fourth to the tenth day. After the tenth day recovery is to be expected.

#### ANALYSIS OF SYMPTOMS

**Diarrhœa**—The watery, pale stools of cholera are alkaline in reaction, have a specific gravity of 1005 to 1010. The dried deposit from a pint of cholera dejections was found by Parkes to weigh only 4 grams. Examined microscopically, the stools are found to contain intestinal epithelium, disassociated or in small flakes, with granular matter resulting perhaps from the disintegration of epithelium along with mucous corpuscles. Before the period of reaction the stools will be found to contain flakes of mucus containing coninas in almost pure cultivation. In the reaction stage these are less readily to be detected, other micro-organisms then abound, and blood cells, not common in the first stage, are now frequently present.

**Chemically**, the rice-water evacuations contain a small quantity of albumin, chloride of sodium, and carbonate of ammonium, occasionally, but very seldom, traces of urea.

The diarrhœa is the primary and constant symptom of cholera. In what has been inappropriately called *cholera sicca*, in which there is neither diarrhœa nor vomiting but rapid collapse, the bowels are after death found distended with rice-water fluid, transuded but not evacuated. The bowel in this dangerous form of the malady is paralysed.

Whether the diarrhœa be caused by the presence of the bacillus in the mucous membrane, abstracting from the blood the liquid necessary for its growth, or by a toxin formed by the bacillus, has not been ascertained.

Diarrhœa is only a symptom, it is true, but it is a symptom producing other symptoms, formidable in itself and in its results. The drain of fluid from the system causes inspiration of the blood, this in turn leads to the absorption of water from all the tissues. Hence the sinking of the eyes, the pinching of the features, the corrugation of the skin of the fingers. This draining off of the watery part of the blood, if not the sole, is an important factor in the arrest of the urinary secretion. The blood does not contain the material for the secretion of urine, but as contributory cause of suppression we must reckon the diminished pressure of the blood in the kidney, from the enfeebled action of the heart. But this cardiac failure is itself, again, partly the result of the thickening of the blood caused by the diarrhœa. The urine is not the only secretion arrested. The secretion of tears, sweat, saliva, and, in most

cases, of milk is arrested. We thus see that on the diarrhœa many of the other symptoms of cholera depend. As a rule, when attempts have been made to remedy the evils caused by the loss of fluids, by means of injection of saline solutions into the veins, the rapid improvement of the patient's condition has shown how essentially dependent many of the prominent symptoms of the disease are on the dehydration of the blood, but the fluids injected in this way are rapidly carried off again by the bowel, and the patient is within a short time in the condition in which he was before the fluid was injected.

**Vomit**—The vomit of cholera is pale or whey-like, alkaline from carbonate of ammonia, and seldom, and only accidentally, contains the comma bacillus. Among the toxic substances isolated from cultivations of the cholera bacillus no emetic principle has been discovered, so far as we know. Yet such a principle is probably present.

**Temperature and Circulation**—The algidity of cholera is to be ascribed to the depressed action of the heart, and also to the thickening of the blood impeding the circulation, especially in the extremities. The depressed action of the heart itself is not the result of a single cause. Brieger has isolated a toxic product of the cholera bacillus which has the effect of lowering the temperature and depressing the heart's action, but it is impossible to doubt that the action of this toxin in producing cardiac depression and lowering of temperature must be increased by the thickened condition of the blood.

Another cholera toxin has been isolated which causes cramps and muscular tremors. We are therefore justified in the present state of knowledge in looking upon the muscular cramps as wholly or in part the result of the cholera toxin.

There is a still more important phenomenon for which we have no satisfactory explanation, namely, the increased temperature of the interior of the abdominal cavity. This appears to be a pretty constant concomitant of algidity. The higher the rectal temperature, the greater the danger. On the approach of death the temperature of the whole body has been found to rise, and it generally continues to rise after death.

A fall of the internal temperature is a favourable sign, and is usually followed by an increase of the surface heat. When reaction is favourable the temperature in the axilla remains normal or moderately elevated. In the typhoid stage it rises above the normal.

**Respiration**—The carbonic acid in the expired air is reduced from the impeded circulation in the capillaries of the lung.

**The Urine**—The causes of the suppression of urine in cholera have been already considered.

#### PROPHYLAXIS

Knowing the sources of the virus, the media

in which it grows, the modes in which it is transported from place to place, and the vehicles by which it is conveyed into the body, prophylactic measures can now be applied with a precision and success that were formerly impossible.

The poison ought to be attacked at its source by the cremation or thorough disinfection of cholera evacuations. In cholera hospitals it will be better to cremate the stools after mixing them with sawdust, but their quantity undoubtedly renders this method of disposing of them difficult in private houses. Carbolic acid in 5 per cent solution is the most useful disinfectant for general use. Bichloride of mercury solution, 1 per 1000, with addition of free hydrochloric acid to prevent the formation of albuminate of mercury, is an effective germicide, but its poisonous properties are an objection to its general use. Chloride of lime, 10 parts in solution to 100 of feces, may also be used. The disinfected stools should be buried at a distance from wells. Clothes may be disinfected by boiling, but as a preliminary they should be fumigated with sulphurous acid. On disinfection by dry heat may be used. Soiled linen and other articles of little value should be burned. Furniture and utensils should be cleansed by washing with carbolic acid, 1 in 20, added to hot water; they should then be exposed to the sun, so as to be thoroughly dried. Rooms and cabins of ships may be disinfected by sulphurous acid or chlorine. For every 1000 cubic feet 1 lb of sulphur is to be used. Chlorine may be evolved by adding hydrochloric acid to chloride of lime in the proportion of 22 lbs of acid to 15 lbs of the chloride for every 1000 cubic feet of air-space. For disinfection of a ship's bilge a 5 per cent solution of carbolic acid, left to act for forty-eight hours, may be employed. We must also mention that Hankin has great faith in permanganate of potash as a means of disinfecting cholera wells. The permanganate is added at sunset, so as to allow the sediment to settle to the bottom. In the morning the water is fit to drink. To our way of thinking, cholera wells should be shut up whenever practicable, and if this is impossible, the water should be boiled before being used for any purpose whatsoever.

The measures demanded for preventing the transport of the disease are the regulation of pilgrimages in India, Mecca, and Mesopotamia. Effective sanitation of pilgrim resorts, means for isolating the sick, arrangements for the inspection of pilgrim ships, their quarantine when necessary, and the disinfection of the clothing and other effects belonging to the infected bands, come under this head.

These are matters of international policy, on which it is unnecessary to dwell. Although strict quarantine is impracticable in a country like England, it is of great value in preventing the introduction of the disease in islands and countries where trade is not extensive and is

limited to a few ports when properly and humanely carried out.

The measures for removing conditions favouring the growth of the germ—what I have spoken of as breeding-places or culture media—resolve themselves into general sanitation. Moist lands should be drained, the pollution of the soil by excreta, and the contamination of the waters of harbours and streams by cholera evacuations, and the discharge of sewage prevented. Cess-pools are to be disinfected and closed, and all refuse and organic matters regularly removed. Gutters and sewers are to be kept in good order. Streets, yards, and houses should be inspected regularly, and measures taken to keep them in a state of perfect cleanliness. If such means be adopted, the germ will find no medium on which to grow, and even should the disease be imported, it is little likely to spread.

Above all, care must be taken to prevent the pollution of drinking water, which is the vehicle for the wholesale diffusion of the virus. No city dependent on shallow wells or on sewage-polluted rivers is safe from cholera outbreaks. An efficient system of filtration (such as that supplied by some of the London Companies) does, however, materially diminish the risk attending the use of sewage-polluted water. When a water-supply has become polluted, the obvious precaution is to boil it before use.

When cholera breaks out in barracks, the troops should be marched out to a dry and elevated locality having a pure water-supply. If the infection persists, the camping ground should be changed. The old rule of marching at right angles to the wind may be safely ignored. The barracks should, in the meantime, be disinfected and the water-supply looked into.

Cholera outbreaks on crowded vessels are always difficult to deal with, and not easy to prevent. Careful daily inspection, the isolation of the first cases, and attention to cleanliness, and, above all, free ventilation are the most likely means to prevent the spread of the malady. If the weather be fine the patients should be put into boats, hung from the davits and fitted up with awnings, and the utmost attention paid to the disinfection of the berths, cabins, and the belongings of the sick.

One other means of prophylaxis which affords a reasonable hope of immunity is the method of vaccination practised by Haffkine. Of 5357 labourers employed in the Cachai tea-gardens, 2381 were inoculated, while 2976 remained unprotected; 4 of the inoculated and 60 of the un inoculated died. In other words, the deaths were proportionately twelve times more numerous among the unprotected.

Unfortunately the protection does not appear to last above a year or a year and a half, yet the value of this method in the case of bodies of men having to live for a limited time in an area where cholera is endemic, or in the case of a

community exposed to an epidemic, cannot be overestimated. If further experience should confirm the reports of the results obtained from these vaccinations, the terrors of cholera will be greatly diminished.

**DIAGNOSIS** — With the aid of a bacteriological examination of the stools, there ought now to be little difficulty in distinguishing severe cases of simple diarrhoea from cholera. A well-marked case of cholera cannot be mistaken for any other disease, except, perhaps, the choleraic form of malarial attack, but attention to the history of the case, combined with a microscopic examination of the blood and feces, will clear up the diagnosis. Poisoning by arsenic, antimony, and corrosive sublimate present many of the features of cholera, but the cholera stools are characteristic.

**PROGNOSIS** — We need not say that in a disease which on an average carries off one-half of those attacked, the prognosis should always be guarded. The disease is most fatal in the extremes of life and in pregnant women. Great prostration, well-marked cyanosis, involuntary discharges, or cessation of the diarrhoea without any sign of reaction, and the occurrence of stupor and persistent anuria after reaction has set in are all of evil augury. On the other hand, a fall of the rectal temperature, an increased fullness of the pulse in the algid state, a normal temperature and absence of typhoid symptoms after reaction has set in, and a urine abundant in quantity and free from albumen are hopeful signs.

**TREATMENT** — Although the indications for treatment are now better understood, we have as yet no means of successfully meeting those indications. In respect to cholera the advice of Hippocrates should never be forgotten, "to do good and do no harm."

The *causal indications* are to destroy the microbe in the intestinal canal or inhibit its growth.

The *symptomatic indications* are to arrest the diarrhoea and vomiting, to restore the fluidity of the blood, to sustain the heart's action, to restore warmth to the external surface of the body, and alleviate the intense suffering caused by the cramps.

In attempting to fulfil the causal indication, crostine, croosote, creole, and salol were tried in Hamburg, but none of them proved of any advantage. Calomel did, however, seem to do good, and the benefit resulting from its use may be ascribed to its germicidal properties. One or two 5-grain doses may be given to begin with, and then 3 grains every six hours. Under the treatment the diarrhoea often augments for several hours and then diminishes. Besides calomel there is no internal remedy that can be safely employed to meet the causal indication, and it must be confessed that calomel does so imperfectly.

Considerable success, however, appears to have followed the use of warm tannic acid enemata recommended by Cantani and others, and, if we mistake not, they were found to be of some service in Hamburg when given in the first stage of the disease. The tannic acid is supposed to destroy the cholera bacillus and neutralise its toxins, and it may do so, but the question is, can tannic enemata reach the seat of the disease? Cantani's formula is Tannic acid, grammes, 3 to 6, gum-arabic, grammes, 30 to 50, laudanum, 30 to 50 drops, infusion of camomile, at 30° C., 1 litre.

**The Diarrhoea** — There is something approaching unanimity as to the advantage of arresting the premonitory diarrhoea. It is believed that the disease may frequently be cut short in this stage by the use of astringents and sedatives, and their employment is also indicated in the first stage of the developed disease. When algidity has set in the diarrhoea usually abates, at any rate absorption is then so completely arrested that this class of remedies would then be useless. Compound chalk powder, opium in combination with sulphuric acid or with acetate of lead, are the remedies most frequently resorted to in order to check the diarrhoea. Graves considered acetate of lead and opium to be the best preparation. He recommended twenty grains of acetate of lead and one grain of opium to be divided in twelve pills, one to be given every half hour until the discharges began to diminish, and he says that "in all cases where medicine promised any chance of relief, this remedy was attended with the very best effects." Perhaps no more reliable remedy to check the diarrhoea can be named than acetate of lead in combination with opium.

**Vomiting** — For the arrest of vomiting ice in morsels, chloroform vapours over the stomach, calomel in large doses — say 15 to 20 grains — mustard emetics, and many other remedies have been tried. None appear to succeed better, or so well, as a tablespoonful of mustard in a pint of warm water. Sometimes a massive dose of calomel will quieten the stomach when other means have failed. In the less severe cases, ice, and a large mustard poultice over the stomach, will prove of service.

**Collapse** — We have seen that the draining off of the water from the blood contributes to the arrest of the secretions, to the enfeeblement of the heart, and the consequent algidity. The injection of saline fluids into the veins has often been followed by a temporary improvement in all the symptoms, but the injected fluid is rapidly discharged by the bowel, and the patient falls back into the hopeless state of collapse from which the injection had for the time rescued him. Some recoveries have followed this plan of treatment, but its employment has generally ended in disappointment, and it is not even devoid of danger. A

safer, but generally an equally unsuccessful, method of supplying the loss of water to the blood is that of injecting water into the subcutaneous tissue. A formula which may be used, if it should be thought proper to try this remedy, is Water, 1000, chloride of sodium, 4, carbonate of sodium, 3. The fluid itself and the instruments must be sterilised. If this is done no harm will ensue, and in a desperate case this remedy might be tried.

With the object of restoring warmth in the state of collapse much benefit will often be obtained from placing the patient in a warm bath. Bögbe found it particularly useful in the early stage of algidity when the patient was young and could be easily moved about. During the epidemic in Naples in 1884, Rigoletti employed the warm bath largely in the algid stage with the best results. The bath was prepared with water at the temperature of 100° to 104° F, to which some liquor ammoniæ was added. Under this treatment the diarrhœa continued, but the vomiting and hiccup ceased, the pulse improved, and the secretion of urine was re-established. This method was also found of service in the recent epidemic at Hamburg, and ought to be tried. In milder cases, hot, moist linseed poultices, sufficiently large to cover the whole abdomen, sides, and lower part of chest, and changed *before* they begin to get cold, or the hot pack, along with friction to the extremities, should be resorted to. If stimulants are to be employed in this stage they should be used in great moderation.

**Muscular Cramps.**—For relief of muscular cramps friction with some stimulating liniment should be employed. If this does not succeed, a tourniquet placed over the upper part of the limb, so as to constrict it, may be tried. The compression should not, however, be continued too long, but having been interrupted, it may if necessary be resorted to again.

**To sum up.** Our efforts must be directed to stop the diarrhœa in the early stage by astringents. If this fail, or if the patient comes under treatment after the disease is developed, calomel by the mouth and tannic acid enemata should be employed. In the state of collapse, the warm bath, hot packs, or, in mild cases, large linseed poultices, with friction to the extremities, and a judicious use of stimulants by the mouth or subcutaneously, are the most hopeful means of relief. The typhoid symptoms which often follow cholera are to be treated on the ordinary principles applicable to the special conditions present.

**Cholera Infantum.** See GASTRO-INTESTINAL DISORDERS OF INFANCY (*Diarrhœa*), COLON, DISEASES OF, DIARRHŒA, FÆCES.

**Cholera Morbus.** See CHOLERA NOSTRAS.

## Cholera Nostras.

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**SYNONYMS.**—*Cholera*, *Diarrhœa*, *Cholera Morbus*, *Cholera*, *Sporadic Cholera*, *Diarrhœa epidemicque* (Fr.), *Sporadische Cholera* (Germ.).

**DEFINITION.**—An acute, intense inflammation of the gastro-intestinal tract, especially of its mucous membrane, accompanied by copious watery discharges from the bowel, and in most instances introduced by acts of vomiting, simulating in the later stages Asiatic cholera, but giving no evidence of the presence of the "comma" bacillus.

**ETIOLOGY.**—The presence of poisonous bodies, animate or inanimate, within the lumen of the alimentary canal supplies the causative agent of choleraic diarrhœa, the resulting effects varying with the precise nature of the poison and the co-morbid environment, both locally and generally. Many agents produce symptoms of diarrhœa physically indistinguishable from one another. The stools in true cholera, occasionally in enteric fever and malarial attacks, and those following arsenical poisoning and apoplexy, apart from any specific micro-organisms in them, are very similar in character to the alvine evacuations of cholera nostras.

In each case the presence of the overting cause leads to inflammation of one, or all, of the coats surrounding the intestine, and frequently of the stomach also.

In choleraic diarrhœa the cause may be organismal, or be derived from the products of organismal metabolism.

Choleraic diarrhœa is, in fact, an inflammation of the digestive canal caused by various agents, but in its results simulating Asiatic cholera more or less closely, absence of the specific "comma" spirillum of the latter disease, and of the symptoms significant of this organism's more intimate and potent influence upon the tissues and processes of the body, serving to distinguish it.

The question of greatest interest for us is concerned with the behaviour and conduct of the bacterial species which normally inhabit the intestinal canal. When of good conduct they serve to control the fermentative processes, brought into being by their own actions, but regulated by reason of the antagonistic properties severally displayed. Commonly peaceable and useful, may they not, stimulated by fortuitous surroundings, acquire and exhibit on occasion a power to do evil? Encouraged by a novel environment, affording them abnormal but more easily assimilable food-stuffs, may

they not wax more rapidly in number, and elaborate more poisonous toxins? Appendicitis would appear often to be due to altered characteristics of this kind endowing micro-organisms in the bowel with harmful tendencies. Intestinal dyspepsia and fermentation, enteritis (but a short step further forward), acute gastro-enteric inflammation can be initiated by similar agents in different stages of virulence. The bacillus coli communis, for example, is a normal peaceful inhabitant of the bowel, usually assisting in the struggle against saprophytic organisms and to check putrefaction, but easily induced, as it seems, to assume new rôles, and, as circumstances favour, to become the active exciting cause of inflammatory conditions both in the bowel itself and in the body tissues.

Bacteria of this class may be supposed not only to increase in vigour and numbers more markedly in the bowel under favourable conditions, or to elaborate more toxic poisons, but to behave in the same manner also if present in food-stuffs before ingestion, and these latter, on gaining the bowel, may be able to work evil.

Whenever gastro-intestinal catarrh occurs, some predisposing cause may be predicated with surety. The exciting cause may come from without, or may be generated within, in either case contributory conditions have nearly always preceded. Insufficient food, or food of unsuitable characters, lack of fresh air, weakened physical and mental powers, exposure to extremes of temperature, particularly if indoors, with overcrowded and non-ventilated rooms, render the chance of an attack more probable and more severe.

The most potent contributory cause of attacks of choleric diarrhœa in babies and sucklings arises from the well-nigh ineradicable belief implanted in the popular mind, that the young were never intended to breathe the fresh air of heaven except during the comparatively brief periods when they are "taken out." Unclean bottles and cups, impure milk, foods unsuited for them at their age, would often prove innocuous, for the body in healthy circumstances is capable of much, if only the child were allowed enough oxygen. Of course, too little food is also a common predisposing cause, and when conjoined to close apartments almost certainly fails to permit of a successful struggle against poisons ingested. No wonder that in enfeebled, marasmic children, hot-house bred, but with less fresh air than is supplied to hot-house plants, starved of oxygen, the bacteria find a congenial soil in which to increase and multiply, little hindrance here to their running amok. Milk that is tainted, so common during summer, especially if the supply is delivered but once in the day, too large a quantity of it given at one time, allowing many of the bacteria in it to escape the antiseptic action of the stomach's secretion, a dietary composed of "bites of whatever is going," want of regulation of the action

of the bowels, constipation relieved at intervals by strong purgatives,—all are active or accessory causes in infants.

It is probable, also, that poisons absorbed into the circulatory fluids of the body, especially those manufactured by abnormal intestinal processes, when in larger amount than can be dealt with by the liver, in virtue of its protective properties, may occasion an attack of choleric diarrhœa by a general in addition to a more local action exercised on the nerve endings and fibrils in the wall of the gut during their transit from bowel to vessel.

The actual and true cause of choleric diarrhœa is almost always to be found in the presence of poisonous products of the metabolism of living forms, and often of the living forms themselves capable of elaborating them. Under ordinary circumstances many of the active agents and some of the poisonous products are unable, by reason of a less activity, a more rapid and thorough neutralisation, to do much harm, but at other times they may be so generated, nourished, maintained, and environed as to possess extraordinary powers of development and increase. Instances of choleric diarrhœa caused by mineral poisons, and many others of the more common chemical substances, fall to be discussed more properly under "Food Poisoning," "Toxicology."

The victims of choleric diarrhœa, if adults, suffer, as a rule, because of their own indiscretion, from the folly of those put over them, if in childhood.

The attacks of the disease are supposed to be more apt to occur under meteorological conditions favouring heat by day with disproportionate cold by night, or when the air is warm and moist. But these conditions can have but little effect on the mode or season of attack unless accompanying ingress of some poisonous agent or substance. And even then those who avoid the inhalation of re-exhaled, impoverished air, nor sleep in the same, long fasts, sluggish action of the bowel, and lack of muscular exertion, seldom are unable to overcome and inhibit the agents introduced into their bowel (unless if in most unusual quantities), which in those who live conversely would be almost certain to find a fertile soil unprotected and uncared for. Impure water, tainted meat, animal ptomaines are more commonly met with in hot weather, in fact, all bacterial life is more active.

**SYMPTOMS.—In Infants.**—The little victim to one of these attacks is in all probability apparently in a normal state of health up to the actual onset of the symptoms. Of a sudden the contents of the stomach are expelled, generally soon after a meal, in an undigested state, and almost at the same time a more or less fluid discharge takes place from the bowel. The discharge is acid, often green or green-yellowish in colour, and containing lumps of undigested

food. In the majority of cases the vomiting soon ceases, but sometimes the stomach may continue irritable throughout. There may be as many as from twenty to thirty motions during the first twenty-four hours, the later discharges becoming colourless, almost odourless, and in fact similar to those of true cholera. The child suffers from thirst, frequently most intense, from abdominal pain, as the drawing of the legs up against the abdomen and crying show, from local pain round the anus, irritated by the acid stools, he becomes prostrated, wizened in look, the body temperature falls below normal, the pulse beat flickers and if the diarrhoea persists, muscular cramps, followed soon by general convulsions, set in, death closing the scene. In severe cases a previously strong, well-nourished child will lose within but a few hours all his healthy curves and plumpness, to show in his appearance all the signs of extreme marasmus.

At any stage of the attack, however, the symptoms may halt and shortly recede, in even seemingly hopeless cases recovery may result (see "Gastro-Intestinal Disorders").

**Adults.**—Few prononitory symptoms assert themselves. Severe purging, with or without precedent emesis or gastric nausea, but always accompanied with paroxysmal abdominal pains, frequently of a most agonising character, the passage first of liquid faeces, dark sometimes in colour, light hue as a rule, irritating and painful the anal surface, of more or less foul odour, becoming more and more liquid, with less and less smell and colour, more frequent and copious, in time indistinguishable from the rice-water stools of true cholera, lead to the sharpened features, the cold, clammy goose-skin, the intense thirst, the lessened urine, the cramps and prostration significant of excessive abstraction of fluid and serum from the organism. The prostration, the cold skin, and the actual lowering of body-temperature which may occur are undoubtedly as often caused by reflex action through the nervous centres from an irritation of the local terminations of intestinal nerves as through actual loss of fluid, while as the resultant effect is to withdraw blood from the skin to supply the inflamed intestinal tissues, the discharge of fluid into the lumen of the gut is facilitated.

Should the attack proceed to a fatal ending, the skin becomes cold and livid, the face more pinched, the muscular spasms more general, although towards the end they are replaced by a comatose, quiet state, or again, the mind may at last retain its full consciousness. During an attack the urine is always scanty, in fatal cases it may be completely suppressed for some time before death.

If the attack arise from ingestion of preformed ptomaines, violently poisonous and irritative in character, the time which elapses between their

ingestion and the onset of the symptoms varies directly with the character of the meal taken which included them, its amount, and the strength of the gastric juice encountered. If the stomach be empty, or nearly so, and the poisonous agent taken in a liquid, the symptoms occur rapidly, if in solid form, a longer time elapses, while if taken along with all the components of a full meal, the onset of diarrhoea may be delayed until eighteen or twenty-four hours have passed.

If the cause be due to organised forms, the appearance of symptoms is usually longer delayed, varying directly with the virulence of the bacterial type and the facilities afforded it for its growth.

One form of acute diarrhoea, in many instances reaching choleraic intensity, is of interest, although of doubtful etiology. It is every now and then met with in large institutions, especially in the writer's experience in boarding schools. From no discoverable cause a number, perhaps even the majority, of the inmates will be attacked almost simultaneously with acute diarrhoea. Seldom serious, much the larger proportion of those attacked recover speedily, a minority, however, may suffer severely. From what can such an attack arise? Not from food, for all eat the same, and all do not suffer.

**PATHOLOGY.**—The pathological conditions are akin to those of acute gastro-enteritis. The mucous lining of the gastro-intestinal wall is acutely inflamed over a more or less extensive area. If the irritant cause is very powerful, the inflammatory changes may spread to the other coats of the bowel wall.

Investigation of the watery stools shows them to be almost entirely composed of pure serum, drained by the intestinal glands from the blood. From them various forms of bacteria can be grown, and ptomaines may be isolated by appropriate methods. The bacillus coli communis is constantly, well-nigh invariably, to be found.

**DIAGNOSIS.**—The most important point in the diagnosis of choleraic diarrhoea lies in its differentiation from Asiatic cholera. This point settled, it is of little moment to be able to distinguish choleraic diarrhoea due to one special form of poison from that caused by another, the symptoms are practically identical, the treatment the same.

A history of previous contact with infection, early onset of collapse, of loss of voice and enfeebled pulse, and the detection of the comma bacillus in the stools suffice to indicate the presence of true cholera.

Occasionally purely nervous diarrhoeic attacks closely simulate choleraic diarrhoea, but here a history of former illnesses of the same type, of some nerve disturbance, the comparatively slight discomfort or pain experienced, the character of the dejecta, less offensive and maybe watery from the commencement, and the slower

onset of signs of exhaustion, serve to point a difference.

The occurrence of choleraic diarrhoea during the progress of enteric fever is infrequent, and can scarcely be mistaken for other than a complication of the primary disease, except when accompanying an ambulatory unsuspected case.

The liquid stools common in intestinal fermentative dyspepsia chiefly call for evacuation in the morning hours, are intensely foetid and feculent, and accompanied by much flatulence.

The symptoms attendant on colitis, mucous and membranous, and the distinctive characters of the motions in these conditions, as well as the differential signs of dysentery and dysenteric diarrhoea, are treated of under their several headings.

**PROGNOSIS.**—Unless among infants, old people with weakened power of resistance or with feeble hearts, or in chronic invalids of little vitality, choleraic diarrhoea, when independent of ptomaine poisoning, is seldom fatal, although seeming on many occasions to nearly terminate death. In infants fed improperly, housed in badly ventilated or unventilated rooms, it proves very fatal. In adults, when uncomplicated, and where proper chances for treatment are obtainable, it rarely causes death, unless it be the result of poisoning by ptomaines taken with the food in sufficient quantity to overpower all means of successful resistance or treatment.

**TREATMENT.**—Starvation is the best line of treatment in both infantile and adult choleraic diarrhoea, cold or iced water, and in adults, aerated water, if desired, if well tolerated, a little milk and lime water may be given, rest the affected parts. In such cases one or even two days may easily pass without nourishment being taken. In infants, unless the diarrhoea ceases early, this should be a constant rule, ignore the protests of parents or relatives. In adults when a desire for food returns, apart from the simple craving for fluids, milk diet may be safely begun.

In adults the less medicine given the better. Cold or iced water, hot-water bottles to the feet and legs, hot poultices, with or without mustard, over the abdomen, and plenty of fresh air generally suffice to arrest the symptoms. The administration of opium or morphia is of questionable propriety. The poison in the alimentary tract merits removal rather than detention, but if the pain be great, and the reflex nervous prostration pronounced, free use of opium will help to remove any chance of immediate collapse. If the patient be in a less prostrate state, the application of warmth, a small dose of calomel—large doses of calomel are dangerous, while small amounts are better fitted to act antiseptically,—and half-ounce or even ounce doses of whisky or brandy, often arrest the symptoms. If painful cramps supervene, etherisation is advisable, indeed the

calmative effect of an anæsthetic upon the whole nervous system, and the relief obtained from the pain of cramp and from the local abdominal pain, suggest that an earlier employment of an anæsthetic in such cases might prove of great service. If seen early, administration of castor oil and laudanum together act well.

Should the stomach be roitive and reject the stimulants ordered, or the patient continue to retch, dilute hydrocyanic acid may be given, or teaspoonfuls of brandy and water, in which one drachm of the spirit is added to two or three ounces of water, the dose to be repeated every few minutes if vomiting threaten. Should the diarrhoea be unchecked by such measures, the sedative powers of opium or morphia may be called upon. The most efficacious method undoubtedly lies in the use of morphia suppositories.

A day or two after the active symptoms have abated, especially if they have been arrested by the use of opium preparations, a full dose of castor oil may be given with safety to clear out all that remains in the bowel.

The general indications for treatment may be summed up in a few words. If the patient appears to be strong enough to bear it, remove the exciting cause, if collapsed, and in danger from persisting exhausting discharges from the bowel, stimulate, and introduce morphia in a suppository or under the skin, in all cases apply warmth to the abdomen and the lower limbs, stop all food by the mouth,—in adults milk may be allowed,—institute rest, and secure the presence of fresh air.

**Cholerine.**—This name (*cholerine*) is sometimes regarded as a synonym of *cholera nostras* (*qv*), sometimes as a term for a form of influenza with intestinal symptoms, or for the milder varieties of diarrhoea which are common during the prevalence of epidemic cholera.

**Cholesteatoma.**—A cystic tumour, with a pearl-like appearance, containing white fat and cholesterol crystals, and sometimes hairs. See BRAIN, TUMOURS OF (*Mixed Anatomy*), EAR, AFFECTIONS OF TYMPANIC MEMBRANE, EAR, MIDDLE, CHRONIC SUPPURATION (*Cholesteatoma*).

**Cholestæramia.**—A group of symptoms (jaundice, irritability, and other nervous symptoms) believed to be due to the presence in the blood of cholesterol. See JAUNDICE.

**Cholesteroline.**—In chemical composition cholesteroline is an alcohol ( $C_{26}H_{48}OH$ ), but it resembles the fats, being soluble in alcohol or ether, its crystals are square with a notch at one corner "Lanoline," or *Adeps Lævis Hydrous*, is cholesteroline fat which has absorbed much water. See EXPECTORATION (*Crystals*); FÆCES (*Chemical Examination*), FLUIDS, EXAM-



INATION OF PATHOLOGICAL (*Chemical Analysis*), GALL-BLADDER AND BILE-DUCTS, DISEASES OF (*Cholelithiasis*); KIDNEY, SURGICAL AFFECTIONS OF (*Cysts*), LIVER, PHYSIOLOGY OF (*Bile Formation*), PHYSIOLOGY, SKIN, ANATOMY AND PHYSIOLOGY (*Secretion, Sweat, Sebium*), URINE, PATHOLOGICAL CHANGES IN (*Cholesterine, Calculi*)

**Cholesteritis.**—A morbid state of the vitreous or aqueous humour in which cholesterol crystals (separated from the lens) are found floating therein

**Cholic Acid.**—Cholic or cholic acid ( $C_{24}H_{40}O_6$ ), present in the bile in the form of taurocholates and glycocholates See PHYSIOLOGY (*Bile*)

**Choline.** An alkaloid ( $C_5H_{15}NO_2$ ) found in bile, in nerve tissue (in lecithin), and in fungi, it is toxic, resembling muscarine in its action, and has been regarded as the cause of epileptic seizures See PHYSIOLOGY, THE TISSUES (*Nerve*)

**Chologen.**—A preparation used in cases of obstruction of the bile-duct, diabetes mellitus, etc

**Cholopolesis.**—The formation of bile See PHYSIOLOGY, FOOD AND DIGESTION (*Bile*)

**Choluria.**—The presence of bile or its elements (*e.g.* pigment) in the urine (*q.v.*), detectable by Gmelin's test (play of colour) See JAUNDICE

**Chondr.**—In compound words *chondri-* and *chondro-* mean relating to cartilage, as is exemplified in such words as *chondralgia* (pain in a cartilage), *chondroarthrosis* (displacement of an articular cartilage), *chondrification* (transformation into cartilage), *chondritis* (inflammation of cartilage), *chondroblast* (a cartilage-forming cell), *chondroblast* (a cartilage-absorbing cell), *chondrocranium* (the early stage of cranial development), *chondrodialysis* (decomposition of cartilage), *chondrogenesis* (formation of cartilage), *chondromalacia* (softening of cartilage), *chondrophyte* (an outgrowth from, or tumour of, cartilage), and *chondrosarcoma* (a variety of sarcoma)

**Chondro - arthritis.** — Guminatous ulceration causing removal of the articular end of a bone, and leaving but a thin membrane (representing the cartilage) over the ulcerated surface See SYPHILIS (*Tertiary, Bones and Joints*)

**Chondrodystrophia Fœtalis.** See ACHONDROPLASIA, CRETINISM, PREGNANCY, INTRA-UTERINE DISEASES (*Fœtal Bone Diseases, Osteogenesis Imperfecta*)

**Chondroma.**—A cartilaginous tumour See BONE, DISEASES OF (*Tumours, Chondroma*), VOL. II

HAND (*Tumours*), HIP-JOINT, DISEASES OF (*Tumours*), NECK, REGION OF (*Solid Tumours*), PAROTID GLAND, DISORDERS OF (*Tumours*), TUMOURS (*Connective Tissue Tumours, Chondromata*)

**Chopart's Operation.** See AMPUTATIONS (*Ankle-Joint*).

**Chorda.**—A cord or cord-like structure, such as the notochord (*chorda dorsalis*), umbilical cord (*chorda umbilicalis*), the *chorda tympani*, and the *chorda tendineæ*

**Chordee.**—A painful erection of the penis, during which it is bent or twisted downwards See PENIS, SURGICAL AFFECTIONS OF (*Chordee*), URETHRA, DISEASES OF (*Gonorrhœa in Men*)

**Chorditis.**—Inflammation of a cord (*e.g.* vocal cord) or cord-like structure

**Chordoma.**—A tumour consisting of notochordal tissue

## Chorea.

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THE name "chorea" has unfortunately been applied to a number of entirely different complaints, the common feature in all "choreic" diseases being the occurrence of involuntary, irregular muscular movements

## COMMON CHOREA

(Chorea Minor, Sydenham's Chorea, St Vitus's

Dance) consists in a group of characteristic symptoms of subacute onset, and chiefly occurring in children.

**ETIOLOGY.—Age.**—Chorea is essentially a disease of childhood and adolescence, the vast majority of cases occurring between the ages of five and fifteen years. After fifteen years of age chorea is much less common as a primary attack, although second and third attacks are by no means uncommon after puberty. After the age of twenty chorea becomes still less frequent, although no age is exempt, and the disease may develop even up to an advanced age, as in one form of *chorea senilis*.

**Sex.**—The female sex is much more liable to this disease than the male, the average proportion being three girls to one boy. After the age of fifteen there is a still greater preponderance of female patients.

**Hereditary Influence.**—A family diathesis towards rheumatism is very common, and should be inquired for in every case of chorea. In a considerable proportion of cases a family history of nervous diseases can also be obtained.

**Rheumatism and Endocarditis.**—Experience shows that a very large number of cases occur in patients who have had articular rheumatism. Many patients develop rheumatism in the course of an attack of chorea or at a subsequent period. Also many cases of chorea are preceded or followed by endocarditis. The endocarditis is sometimes discovered for the first time during the course of the chorea, and it is rare to find a patient with a second or third attack of chorea in whom the cardiac sounds are normal.

In a recent series of forty-one consecutive cases of chorea, in twelve of them (including a primigravida) there was a history of previous rheumatic fever, another patient developed pain and swelling in both wrist-joints whilst under observation, within a week after the onset of the chorea, whilst another primigravida, who had previously had two attacks of chorea, developed pain and swelling in one knee-joint a fortnight after the commencement of her attack of chorea gravidarum. Of the remaining twenty-seven cases, fifteen had a strong family history of rheumatic fever, whilst in the remaining twelve there was no history of rheumatic fever either in the patient or in the patient's near relations. Yet out of these twelve no fewer than five had mitral regurgitation, one had mitral stenosis which ultimately proved fatal, and only six had no valvular affection of the heart.

Scarlet fever with arthritic manifestations is recorded in a few cases to have been a direct antecedent of chorea.

**Fright, emotion, traumatism,** or some variety of shock to the nervous system is a fairly common antecedent to an attack of chorea, although the majority of cases develop without such an exciting cause. Still a history of some fright or shock is obtained in from 20 to 30 per cent of

cases. Mental emotion as an alleged exciting cause is more common in adolescents, from the age of sixteen upwards, than in childhood. But the study of a number of cases of chorea attributed to emotion shows that the vast majority of them occur in constitutions which are already rheumatic. Moreover, the history of mental shock is often an indefinite one, and early choreic symptoms are often present before the advent of the emotion which exaggerated them. In the above series of forty-one cases, fifteen gave a history of some fright or mental shock, but of these fifteen only one was free from evidences of rheumatism, twelve of the others having either had rheumatic fever or mitral disease, and the remaining two having a very strong family history of rheumatic fever.

It used to be thought that chorea may spread from one patient to another by *imitation*. Small epidemics of choreiform movements, such as have now and then been recorded in girls' boarding-schools, are really hysterical in nature and not truly choreic. A primarily choreic child, however, may have a relapse of true chorea from the excitement of seeing another patient with a similar affection.

The presence of *worms* in the intestinal canal has been believed by some to cause the disease in certain cases by reflex irritation. But in the above series of cases the only case of chorea in a child with tape-worm occurred in a girl who had previously had an attack of rheumatic fever.

**Pregnancy** undoubtedly acts as an exciting cause in certain patients, the disease developing in the early months of gestation, usually from the third to the fifth month, either in previously healthy patients, or more frequently in cases with a previous history of rheumatism or of actual chorea. Young primigravidae are chiefly so affected, frequently unmarried girls. The connection between the chorea and the pregnancy is so close a one that when the pregnancy comes to an end, either in a normal manner or by abortion, the chorea ceases. Frequently the disease recurs with succeeding pregnancies (*vide* "Chorea Gravidarum").

**SYMPTOMS.**—The disease is gradual in onset, usually taking a day or two to develop sufficiently to call attention to the presence of any abnormality. The patient is often fretful and more irritable than usual, then restless, wiggling movements are observed in the limbs, face, and trunk. The child at school cannot sit still, and the teacher complains that the patient's handwriting is clumsy and untidy. Often the patient drops things suddenly out of the hand. Sometimes this condition is not recognised as being really due to disease, since a degree of motor restlessness is present in many children during mental embarrassment or other emotion. The choreic child is often considered at first to be merely badly behaved, but as the symptoms

develop, it becomes evident that there are some underlying diseased conditions.

In a typical, fully developed case of chorea the symptoms may be classified into various groups, viz (a) irregular, involuntary movements, (b) inco-ordination on attempted voluntary movements, (c) weakness of the affected limbs, (d) a variable amount of psychical disturbance.

The irregular, involuntary movements are quite characteristic. They are of a twisting, wriggling, tossing type, quite irregular in time, and constantly varying in character and degree. The patient cannot stand or sit still, but is in a state of continual motor restlessness—"fidgeting." The movements are usually first noticed in the arm or face, the legs, as a rule, being less affected. The shoulder may be suddenly shrugged, the arm abducted or adducted, the elbow flexed or extended, the hand pronated or supinated, the fingers spread out, flexed, or extended, and these sudden irregular movements are often combined in a complex, confused fashion.

Similar movements are also seen in the face. The brow may be suddenly wrinkled, or brief grimaces may be made, the mouth being drawn to one side, the lower lip everted, the mouth pursed up, the upper teeth exposed, or the eyes screwed up. The ocular muscles also participate; the patient does not fix objects steadily, but looks restlessly hither and thither, and may now and then squint momentarily. The jaws may open and shut irregularly, the tongue may be rolled about in various directions, and not infrequently it is bitten by a sudden involuntary snap of the teeth. Sometimes the soft palate is seen to move irregularly up and down. The movements of the muscles in and around the mouth may produce peculiar sucking, snacking, or slobbering noises.

The trunk is often rotated to one side or the other, and the head suddenly turned in various directions. Respiration is frequently jerky and irregular. The action of the diaphragm is often overshadowed by that of the intercostals and of the extra muscles of respiration. Sometimes the diaphragm and intercostals contract alternately instead of synchronously. In a few cases the laryngeal muscles are affected, so that jerky laryngeal noises or groans are produced.

Choreic movements in the legs are usually of a much less complicated type than in the upper extremities. The patient cannot stand placidly, sometimes one foot is moved, sometimes the other. Walking is only impaired in severe cases. The knees may suddenly give way during progression, or the movements of the legs may be so violent that the patient is unable even to stand.

The choreic movements vary in degree in different cases, from the slightest restlessness of the fingers to the wildest and most violent, irregular movements of all the voluntary muscles of the body. The affected joints are impetuously

flexed, extended, rotated, or circumducted, and in severe cases the limbs and trunk may be thrown about so violently that the patient may fall out of bed, or may bruise or cut the bony prominences of the head, trunk, or limbs by throwing them against surrounding objects.

The movements usually commence in the hand, spreading later to the face, trunk, and leg. They generally affect one side of the body earlier and more severely than the other. Often they remain confined entirely to one side (*hemi-chorea*). In a few cases they migrate completely from one side to the other. As a rule, in right-handed patients the left side is more commonly affected than the right, but this is not invariably the case. Sometimes the movements afflict both sides with equal intensity.

Voluntary movements are more or less interfered with, when involuntary choreic movements are going on. The patient executes voluntary movements hastily and spasmodically as if attempting to seize an instant when choreic movements are absent. Sometimes, however, voluntary movements are almost steady, whilst involuntary movements are well marked, but more commonly choreic movements are increased by voluntary movement. A good test for slight cases of chorea is to make the patient hold up both hands for a few seconds above his head with the fingers outstretched. This usually succeeds in bringing out a few irregular, wriggling movements of the fingers on the affected side. Or make the choreic patient squeeze one's hand, and the grasp is perceived to be sudden, spasmodic, and ill-sustained. If the grasps be compared on the two sides in a case of *hemi-chorea*, it will be found that the grasp on the choreic side, although more sudden, is yet actually weaker than on the unaffected side. Sometimes when picking up objects the patient makes a sudden dash for the article and often drops it after securing it. Sometimes, again, the patient has a difficulty in letting go an object when he wishes to do so. Thus in feeding himself he often drops his cup, and scatters his food about the table with his knife and fork.

In the lower limbs, in addition to the involuntary movements already described, the gait may be peculiarly altered. The knees may suddenly give way during walking, causing the patient to fall.

When the tongue is protruded it is shot out suddenly and pulled in again with a jerk. Articulation is often jerky and indistinct. Words are shot out, suddenly cut short, or interrupted by the irregular respiratory movements. In bad cases the speech may be quite unintelligible, or the patient may speak only in whispers, or may even absolutely refuse to speak at all for days or weeks. In the latter instance there is a superadded psychical element present.

Severe affection of the lips, tongue, and pharynx may cause inco-ordination of swallow-

ing, and the general nutrition may thereby be gravely affected

The motor unrest is greatly increased by emotional states. The excitement of a medical examination often renders the movements much more violent. Scolding the patient by teachers or parents only makes the child more restless. Sometimes the patient sleeps badly. As a rule, during sleep the choreic movements entirely cease, but in very rare cases exactly the reverse condition is found (*chorea nocturna*), in which the movements are greatest during sleep, disappearing almost completely when the patient is awake.

A considerable degree of muscular weakness is common in chorea, and in some cases this may be so severe that the patient is unable to raise a limb from the bed, although slight restless movements are seen going on irregularly in the weakened limbs. But the patient can always execute voluntary movements, however feebly, at all joints in the apparently paralysed limbs. There is never complete paralysis of any group of muscles. Such cases, where the weakness is out of all proportion to the choreic movements, have been termed "paralytic chorea," and in many of them the choreic movements are only elicited on attempted voluntary movement. In some cases muscular paresis is the earliest symptom of the disease. The patient's relations notice that the child uses one arm less than the other, and finally that he does not use it at all. As the disease recovers the paresis fades, and the choreic movements become more evident.

The muscles remain normal in volume. Their electrical excitability is sometimes increased on the affected side, both to the continuous current and to induction shocks.

Sensory abnormalities are more frequent in chorea than it is generally supposed. It is true that choreic patients do not usually complain of pain, numbness, or any abnormal feeling, but if the cutaneous sensibility be carefully tested in every case, it will be found that a considerable proportion of patients exhibit a slight degree of blunting of sensation on one side of the body. This was so in ten cases out of forty-one in the above series. Such hemi-anesthesia, when present, is always on that side of the body on which the choreic movements are most marked. Sometimes the visual fields are concentrically contracted, and the acuity of vision, smell, taste, and hearing may be diminished on the preponderatingly choreic side, with or without slight hemi-anesthesia to touch or pain. Such affection of the special senses is strongly suggestive of a hysterical element superadded to the chorea, but the frequency of hemi-anesthesia is very striking. The hemi-anesthesia of chorea is very slight in degree, and can only be detected by careful comparison of corresponding points on opposite

sides of the body. When this is tried a considerable proportion of patients will be found to perceive light touches and pricks more acutely on one side than on the other, and the boundary of this area of blunted sensibility will always be found to lie in the middle line of the body. Sometimes the trunk and limbs are alone involved in the hemi-anesthesia, the face and scalp escaping.

As to the reflexes, the skin-reflexes are occasionally diminished on the hemi-anesthetic side. The deep reflexes may be normal, but sometimes they are difficult to elicit. In other cases we may find the "choreic knee-jerk." This differs from the healthy knee-jerk in the fact that when the patellar tendon is tapped, instead of the normal brisk contraction of the quadriceps extensor, followed at once by a sudden relaxation, the knee in such choreic cases is suddenly extended to its full extent and remains so for a second or two, the foot and toes meanwhile exhibiting irregular choreic movements. Ordinarily the sphincters are normal, except in very bad cases when extreme mental dullness causes incontinence of urine and feces.

In chorea the optic discs are normal. In rare instances slight optic neuritis has been observed. This is probably not referable to the chorea itself, but due to some other concomitant cause. The pupils are often dilated, but they react normally. The temperature is normal, except in very severe cases, when it may rise.

In the majority of cases a psychical element is present. This may be trivial in degree, merely amounting to a certain irritability of temper or a tendency to cry or to giggle on slight provocation. Sometimes, however, there is impairment of memory and distinct mental dullness. The mental element may be quite out of proportion to the severity of the other symptoms. Mental affection is more common in adolescents than in young children. It usually takes the form of depression. Hallucinations may develop with delirium, and the patient may pass into a state of violent maniacal excitement. This maniacal form (*chorea insaniens*) reaches its maximum intensity at the acme of the chorea. It may last several weeks, and generally passes ultimately into a condition of mental apathy in which the patient lies in bed like a log, refuses food, and passes both urine and feces into the bed. As the patient slowly recovers, there is often a transient stage of mild delirium with "persistent ideas." Severe mental symptoms are more common in the chorea of pregnancy than in any other variety of chorea. The ultimate prognosis, however, as a rule is good, most cases of choreic insanity recover completely.

The urine in chorea contains an excessive amount of urea, of phosphates, and sometimes also of hematoporphyrin.

COMPLICATIONS.—Of these the most important is endocarditis, which occurs so frequently that

it may almost be considered as part of the disease. Choric endocarditis chiefly affects the mitral orifice and is usually of the simple type. The heart frequently recovers completely and its bruits entirely disappear. Sometimes, however, the valves may remain permanently damaged. Ulcerative endocarditis with embolism is rare.

Acute articular rheumatism may precede, accompany, or follow an attack of chorea. When occurring simultaneously with chorea, it is usually mild in type, evidenced chiefly by joint pains and moderate pyrexia, with little or no joint swelling to be made out on objective examination.

Some choreic children have characteristic "subcutaneous rheumatic nodules," varying from the size of a pea downwards to that of a sago-grain. They are distributed most commonly along the subcutaneous borders of the ulna and tibia, also around the elbow, knee, and ankle joints, and along various tendons around the wrists and ankles. Sometimes these nodules are only discoverable during the attack of chorea. Erythema nodosum is sometimes present. A painful contracture of the palmar fascia may also occur during chorea, clearing up under anti-rheumatic treatment.

Cases of chorea with intense mental symptoms have already been referred to. Such maniacal cases occur chiefly in adults, and especially in the pregnant state.

Epilepsy as a complication is very rare. Hysteria may complicate chorea, and hysterical movements may sometimes simulate chorea, but the practised observer has usually little difficulty in recognising the existence of a hysterical element when present. Hysterical movements are usually somewhat different in type from those of true chorea; they are often sudden and shock-like, and tend to be more rhythmical than in true chorea.

**DURATION.**—The duration of an attack of chorea is very variable. The more severe the attack, the longer will be its probable duration. The average duration is from two to three months, but an attack may clear up in two or three weeks, or it may last six months or longer. Only in exceptional cases does chorea persist continuously for a year or more.

As a rule, the older the patient is when first affected by chorea, the longer is the duration of the attack likely to be.

Chorea is a disease which is very prone to recur. One or two relapses are common, and as many as nine relapses have been recorded. The intervals between two successive attacks vary from a few weeks to several years, the average interval is about one year. In some cases the patient has an attack of chorea regularly at a certain season of the year for several successive years, but as a rule the attacks recur at quite irregular intervals.

Relapses are often slighter in severity than the primary attack, but the reverse is sometimes the case.

**PROGNOSIS.**—In most cases recovery is complete, the inco-ordination gradually ceases and at last the involuntary movements fade away. Serious cases are those in which the movements are of extreme violence, and in which insomnia, delirium, and rapid emaciation supervene. Those may end fatally from exhaustion. Sometimes death results from rheumatic fever with hyperpyrexia, or from cardiac failure secondary to valvular disease. Injuries to the skin produced by the violence of the choreic movements may result in intractable sores, and the patient may die from pyæmic infection.

In the common chorea of childhood the danger to life is but slight, the average mortality being less than three per cent. Most of the fatal cases are first attacks, death from a recurrence is rare. Chorea occurring for the first time at or after puberty is much more dangerous, since severe heart-lesions are more frequent than in the chorea of childhood.

Chorea in pregnancy is still more serious, nearly twenty-five per cent of cases proving fatal. In such cases the choreic movements are usually very violent, mental complications are common, and severe endocarditis is relatively frequent. Many cases abort, and in the patient's cachectic state abortion or even normal labour is dangerous.

In extremely rare cases juvenile chorea may persist as a permanent chronic disease, with or without slight remissions, in spite of all treatment. This form of chorea is commoner in males than in females, and seems to be less definitely associated with rheumatism and endocarditis than ordinary chorea is (*vide infra*, "Chronic Progressive Chorea").

Chorea developing after middle life is much more likely to be a permanent disease than the juvenile form.

**PATHOLOGY.**—Chorea has a clinical rather than a pathological existence, and the disease is at present classed amongst "functional" disorders. Practically nothing is known of its essential pathology. It is only in severe and complicated cases that the disease is fatal, and at post-mortem examinations very varied and inconstant appearances have been found, amongst which may be mentioned general hyperæmia of the brain, embolism or thrombosis of cerebral vessels, and minute perivascular hemorrhages in the brain and spinal cord. Sometimes fine changes in the nerve-cells of the central ganglia have also been described. But an insurmountable objection to the acceptance of any one of the above as the essential lesion of the disease lies in the fact that any or all of these changes may be absent and the results of examination may be negative, the brain, spinal cord, and peripheral nerves appearing absolutely normal.

—so far, at least, as the present methods of neuro-pathological research enable us to judge recent endocarditis, however, is found in a great majority of fatal cases of chorea.

Some time ago the disease was supposed by some to be due to extensive capillary embolism, and this view was thought to be strengthened by the fact that the injection of starch granules into the carotid arteries in dogs produces choreoid movements. But most cases of fatal chorea show no signs whatever of such embolism. Moreover, "canine chorea" is essentially a different disease from chorea in the human subject.

The nature of the choreic movements, together with their cessation during sleep, points strongly to their origin from some irritable condition of the motor cells of the cerebral cortex. Such a hypothesis is further strengthened by the frequency with which the symptoms are confined to one side and by the frequent presence of mental disturbance.

For want of a better explanation, chorea is at present classified as a "functional" disorder of the nerve-cells. But there can be little doubt that some molecular or chemical change in the neuron underlies all so-called "functional" disorders, though as yet we are ignorant of the precise nature of such changes.

It is not at all improbable that chorea in many cases may be due to a toxin, probably closely associated with the rheumatic poison, if not identical with it.

That emotion frequently plays a part as an exciting cause is not antagonistic to such a theory, for the mental shock in such cases often merely precipitates an attack in an unstable or imperfectly developed nervous system already partially poisoned by a toxin in the blood. Endocarditis is as frequent in cases which have followed upon mental emotion as in those which have arisen spontaneously, and yet no one doubts that endocarditis is due to the existence of a poison in the blood. Endocarditis is so common in chorea that we conclude there must be some close connection between the two, and the fact that chorea so often precedes the endocarditis shows that the latter cannot be the cause of the chorea. We are therefore again driven to the conclusion that some common cause must bring about both the chorea and the endocarditis.

**DIAGNOSIS.**—Ordinarily chorea can be diagnosed at a glance. The peculiar movements are quite characteristic, and the history of a recent onset distinguishes them at once from athetosis following cerebral palsy in early life, which is the commonest condition in which similar movements occur. The movements of athetosis have unfortunately been termed "post-hemiplegic chorea," but the diagnosis between the two conditions is usually easily made, not only from the "athetoid" character of the movements, but also from the presence of

muscular rigidity in the athetotic limbs, with a history of some previous cerebral attack (*vide* "Athetosis," vol. 1 p. 319).

The rare condition called General Convulsive Tic ("Tic Générale") simulates chorea even more closely, but can be differentiated by observing the systematic nature of the movements in Convulsive Tic, the relatively long intervals of rest between the movements, the fact that the patient can always intermit them in order to execute voluntary actions, and the frequent presence of echolalia, coprolalia, or other explosive utterances and of forced movements.

Sometimes difficulty arises in the diagnosis of cases of "paralytic" chorea, where the outstanding symptom is loss of power in one arm. When a child between seven and twelve years of age is said to have gradually lost the use of one arm, without paralysis of leg or face, the disease, as a rule, is chorea. Careful observation in such cases never fails to reveal slight choreic movements in the affected limb.

The few cases of chorea where the legs are chiefly affected may simulate ataxia of gait, but the choreic movements of the limbs in the sitting and recumbent posture are sufficient to differentiate the two conditions.

**TREATMENT.**—The mental and motor symptoms of chorea, together with the marked influence which emotion and exertion, mental or physical, have in exaggerating the choreic movements, make it obvious that a most important element in the treatment of chorea consists in the removal of all causes of emotional excitement and physical or mental fatigue.

The patient must not go to school. Lessons must be stopped at once and rest in bed should be insisted on, for several weeks at least, even in mild cases. When the patient is afterwards allowed up, this should only be for a short time each day, gradually lengthening the time spent out of bed.

All sources of mental irritation must be avoided, and only such occupations should be permitted which interest the patient without fatigue or excitement. The child must not be frightened or threatened. A large airy room, if available, should be chosen for a bedroom, and cheerful companionship should be provided, such as that of the mother or of a judicious nurse, since mental depression might undo the good produced by rest. If there are other children in the house, they should not be allowed to go into the patient's room, lest they increase his mental and physical unrest.

In cases with violent movements, care must be taken to prevent the patient from falling out of bed or injuring himself. This can be accomplished by laying the patient's mattress on the floor in a corner of the room, well padded with cushions or pillows, to prevent him from knocking himself against the wall. If bed-sores threaten, a water-bed should at once be procured.

Treatment in an asylum is seldom necessary, except in cases with severe mental symptoms where isolation at home is unattainable. But if a case is doing badly at home, transference to hospital is often advantageous and expedites the cure.

The diet should be nutritious and easily absorbed. The more food the patient can take the better. In severe cases where deglutition is unpaired, or where from mental dulness the patient does not take enough nourishment, nasal feeding must be resorted to. Hughlings Jackson gives alcohol—a teaspoonful of brandy every four hours—in severe cases of chorea, and with excellent results. The bowels must be kept open, but strong cathartics are to be avoided.

It is important that the patient should sleep well, and hypnotics must be given if necessary.

As to drugs, arsenic is the classical remedy. It is given by the mouth, and the dose should commence with three or four minims of the liquor arsenicalis three times a day, increasing gradually to ten or fifteen minims. If symptoms of arsenical poisoning appear, such as conjunctivitis, nausea, and gastric pain, the drug must be stopped for a few days and then resumed in a somewhat smaller dose. The administration of large doses of liquor arsenicalis (fifteen to twenty minims three times a day) from the first, for a period not longer than a week, has sometimes yielded good results, but if this mode of administration be persisted in for a long period, it is not devoid of risk. Arsenic in large doses administered for a lengthened period not only induces a brownish pigmentation of the skin, but has sometimes produced very intractable peripheral neuritis.

In severe cases chloral hydrate is of great service as a hypnotic and general nervous sedative. Some physicians keep the patient continuously asleep for weeks by means of this drug, but caution is necessary in such cases, since the chloral sleep is sometimes followed by maniacal excitement.

Bromides are of singularly little value in chorea. In very violent cases morphia may be administered hypodermically, or it may even be necessary to give chloroform to control the violence of the movements, but such treatment can only be of temporary service, it being much better to keep the patient under the influence of chloral. In some cases antipyrin is useful, but care must be taken not to produce cardiac failure, therefore during treatment by antipyrin the patient must be kept absolutely at rest.

Valerianate of zinc, oxide of zinc, hyoscyne, cannabis indica, and physostigmine have all been employed, but are of little use. Salicyl compounds occasionally give good results, especially salol, but in other cases, again, they fail entirely.

The application to the spine of electricity,

faradic or galvanic, and the freezing of the skin over the spine by means of ether spray, which have sometimes been employed, are of doubtful value, and when we remember that the disease is of cerebral and not of spinal origin, this can readily be understood.

During convalescence mild gymnastics are often of service, and a change of air often serves to confirm the cure.

Finally, it must be remembered that chorea is a disease which is prone to recur. Each successive attack must be treated on the same principles, rest and feeding being more important than medicinal therapeutics.

#### HEREDITARY ADULT CHOREA (Huntington's Chorea)

This is a somewhat rare disease which must be carefully distinguished from the ordinary chorea minor.

The disease was described in 1872 by Huntington of New York as a form of chronic progressive chorea occurring hereditarily in certain families through many generations, the malady commencing in middle life, being associated with mental weakness, and continuing until death. Since its original description the disease has also been observed with considerable frequency in Britain and on the continent of Europe.

The complaint is markedly hereditary in certain families and has been traced through many successive generations. Sometimes, however, one generation escapes and the disease does not afterwards reappear in the family.

Males and females are equally affected, and both sexes may transmit the disease, which begins, as a rule, in middle life, commonly between the ages of thirty and fifty years.

It may arise without any cause whatever, but sometimes it first becomes evident after emotional excitement. Thus in one woman, whose mother and maternal grandmother had been similarly affected, the earliest symptoms came on after the physical and mental shock of being forcibly discharged by her employer, who threw her down a flight of stairs.

*Symptoms.*—The patient exhibits movements of a choreic type, usually first noticed in the face or upper limbs, gradually increasing in severity, and ultimately, in the course of years, affecting all the voluntary muscles of the body. The motor symptoms are similar to those of chorea minor, with grimacing, gesticulation, affection of articulation, and almost constant play of involuntary irregular movements of the eyes, face, limbs, and trunk. The ocular muscles do not necessarily escape, as some authors state. Sometimes one side of the body is affected for years before the other side is attacked.

The movements, as in chorea minor, cease during sleep and are increased by emotional excitement, but, unlike ordinary chorea, they can be arrested when the patient desires to

execute any voluntary movement, thus the patient can still write or thread a needle, even when the disease is far advanced

Even up to a late stage the patient is able to walk, but the gait is often curiously irregular. The trunk and limbs undergo irregular spontaneous movements, the patient often suddenly standing still after walking a few steps

Sensation is normal. The deep reflexes are generally somewhat increased. The heart is unaffected

Mental weakness is usually present, commencing as mere "futility" and gradually advancing towards a degree of dementia. The patient is often depressed, and suicidal tendencies have sometimes been observed. The patient may at first be irritable or excitable, but, as the disease progresses, the mental state usually becomes one of apathy. The mental affection is an insidious one and generally develops some years later than the muscular restlessness, but this is not invariably the case. Mental weakness may precede the motor phenomena

The disease is an incurable one and its symptoms persist throughout life, but life is not necessarily shortened thereby. The complaint may last from ten to thirty years, and the patient ultimately dies either from exhaustion or from some intercurrent affection

**Diagnosis.**—This disease is distinguished from common chorea chiefly by the history of heredity, which in ordinary chorea is very rare, also by the age of onset, the tendency to dementia, the prolonged duration, and the progressive nature of the malady. Chorea senilis, which may either be chorea minor occurring at an advanced age, or may be a variety of chronic progressive chorea, is free from any hereditary tendency and unassociated with progressive dementia

**Pathology.**—As in chorea minor, the essential pathology of this disease is quite obscure. It is probably a degenerative process affecting the cortical motor neurons. Very various morbid appearances have been found at autopsies, including slight diffuse chronic meningitis, changes similar to those of general paralysis of the insane, pachymeningitis hemorrhagica, disseminated miliary nodules of chronic inflammation scattered in the white and grey matter, etc., but these are all probably accidental, and not essential to the disease

**Treatment** can only be symptomatic. Aisenic is of little or no use. Tonics and various sedatives may be tried, but therapeutic results have, so far, been entirely negative

#### CHRONIC PROGRESSIVE CHOREA

Apart from Huntington's chorea, other forms of chronic progressive chorea occur unassociated with any hereditary tendency. Isolated cases may begin in old age, in adult life, and recently have also been observed in adolescence (*developmental chorea*)

These cases are distinguished from ordinary chorea by their duration and by the absence of rheumatic affections or of cardiac complications. The motor symptoms are exactly those of chorea minor, but they become progressively worse and last throughout the patient's life

Such varieties of chorea are obviously different from ordinary chorea minor, which, though it tends to relapse, ultimately in most cases becomes cured. They are probably dependent on a chronic degenerative process in the cells of the motor cortex, rather than upon any toxin, as in the case of chorea minor

#### ELECTRICAL CHOREA (Dubini's disease)

This is a very rare endemic disease, which occurs chiefly in Northern Italy and Lombardy. It has been observed in both sexes and at all ages. The disease is a progressive one. It commences with a short stage of pains in the head, neck, or spine, soon followed by involuntary spasmodic movements which differ from those of ordinary chorea in being short, sudden, and shock-like, similar to those elicited by electrical stimulation

These contractions usually commence locally in one arm or one side of the face, later they attack the leg on the same side, and at last they cross over and invade the muscles of the opposite side. In addition, there are sometimes epileptiform convulsions, which may be unilateral

As the disease progresses, paralytic phenomena are superadded, commencing in those limbs in which the contractions were earliest and most marked. The palsy, which is accompanied by muscular atrophy and diminution of faradic excitability, spreads all over the body and becomes general. There may be considerable elevation of temperature. Throughout the disease the mind is unaffected

The complaint may last for days, weeks, or months, and is usually fatal. Death results from cardiac failure or from coma. Only in a few cases does recovery take place

The nature of the disease is undetermined. No constant morbid changes have been found. On the whole, it seems probable that the disease is due to some toxin of endemic origin which attacks first the cerebral cortex and secondarily the spinal cord

**Diagnosis.**—This disease has not been observed except in Italy. Elsewhere the only complaints which may simulate electrical chorea are myoclonus multiplex (*q.v.* "Muscles, Diseases of the") and certain varieties of hysterical spasm, both of which conditions have only to be known in order to be readily distinguished from Dubini's disease

The so-called *Chorea major* (*Chorea magna*) must be carefully differentiated from all the truly "choreic" disorders. It is not a chorea at all, but merely a variety of hysteria easily distinguished from true chorea



## CHOREA GRAVIDARUM

Chorea, rare in the adult, is perhaps a little less rare in the pregnant. This is, however, by no means certain. Of 439 cases in the British Medical Association's collective report, 68 were females between 16 and 40, and of these 7 only were pregnant, of 28 delirious female cases, over 15 in the Kommunehospitalet at Copenhagen, 7 were pregnant and 1 recently delivered. The proportions in these instances are 1 in 9 and 1 in 3, while the proportion of pregnant women to adult women in general is between these ratios and nearer the higher. Nevertheless, the association of chorea and pregnancy is important both for prognosis and treatment.

Cases of chorea in pregnancy exhibit the same relations to fright, rheumatism, and endocarditis, hysteria, family tendencies, and previous chorea, as cases of chorea in general. Fifty-nine per cent of first attacks occur in the first pregnancy, but the ratio of illegitimacy is little if at all in excess of that of women in general. Corresponding to this, the age at the first choreic pregnancy is usually under 25, but cases are on record at all ages up to 40. Chorea may recur in several pregnancies, the largest number of recorded attacks being 5, but choreic pregnancy may be preceded or succeeded by pregnancy free from chorea. Fully half of the attacks began in the first three months, and a third in the middle three months. A few cases are on record where chorea began during the puerperium, and two or three somewhat later.

Of cases where the pregnancy ends spontaneously two-thirds are delivered at term, the proportion of abortion being thus higher than in general. In about 40 per cent of the cases that recover the chorea terminates before the end of the pregnancy. There is no evidence to show what influence the death of the fetus, before delivery, has on the chorea, in a few cases the perception of the fetal movements has exaggerated the chorea. Recovery after delivery is, as a rule, gradual, as it is in chorea apart from pregnancy, but in a few recovery is sudden, in one case the chorea became chronic. The mortality cannot be exactly determined, but is certainly less than 1 in 5, and a considerable number of the fatal cases die from causes other than the chorea. In less than 3 per cent of the cases insanity followed the chorea, recovery taking place in a few weeks or months. In a number of cases infective processes have complicated or followed the chorea.

The treatment of chorea in pregnancy is primarily the same as the treatment of chorea generally. Arsenic and salol, and the sedatives and narcotics, including general anaesthesia, must be employed according to the indications of the individual case. Where the case is severe or becomes worse in spite of such treatment, the induction of abortion or of premature labour is

justified. In practice this has not always given satisfactory results, but the cases were of great severity, and sometimes the delay had been too great. The induction should be made under anaesthesia, which may reasonably be prolonged till the completion of the delivery. In some cases digital dilatation of the cervix has stopped the chorea. In less severe cases and those which improve under general treatment, it is best to wait spontaneous labour and to watch that, interfering only if this seem specially required.

## CHOREIC INSANITY

There is probably no special form of insanity that can properly be called Choreic Insanity. In every patient suffering from chorea there is usually some mental disturbance. The patient may be dull and listless with apathy and loss of memory, but these mental symptoms being so slight they are overlooked, on the other hand, however, they may become so severe as to call for immediate treatment.

*Etiology*.—Mental complications with chorea are more common with pregnant women, girls, and young men than with children. Usually there is an unstable inheritance.

*Symptoms*.—1 *Choreic Mania*.—The excitement rarely begins before the end of the first and after the fourth week of the outbreak of the chorea. Often it is most difficult to say when the bounds of sanity have been passed, as the impulsiveness, agitation, or loss of control grow gradually. Sleep becomes more and more interrupted until it is finally lost altogether. The patient becomes destructive and dirty in his habits. Hallucinations of sight and hearing may be present. He is incoherent, is fed with difficulty, and there is steady failure of physical condition.

2 *Agitated Melancholia*.—Patient is depressed with self-accusations, restless, despondent, and believes that he will never recover. There is often persistent refusal of food. This type is more common in young pregnant women.

3 *Acute Delirium*.—These cases differ from those of acute mania by the severity of both the mental and somatic symptoms. The mental condition resembles that of acute delirious mania. There are marked fever, hallucinations of sight, refusal of food, constant excitement, incontinence, great prostration and exhaustion, and the termination is often fatal.

4 *Delusional Insanity*.—Delusions of persecution with the belief that they are being annoyed may develop in cases of chronic chorea.

5 *Stupor*.—The mental hebetude and inattention that is so common in choreic patients may become more profound, and they pass into a stuporose state. The memory is commonly very defective in these cases.

*Morbid Anatomy*.—There are at present no definite macroscopical or microscopical changes

known that can be said to be the actual cause of choreic insanity,

**Prognosis.**—The prognosis is usually good in the mania and agitated melancholia of chorea. With acute delirium the condition is much more serious and the prognosis must be always guarded, but when the patient once begins to improve there is seldom a relapse.

Delusional insanity or stuporose cases are commonly chronic.

**Treatment.**—This is largely upon general lines. Good feeding with large quantities of milk and eggs, custards, and solid food, if possible. The more acute the attack the greater must be the amount of food given. If nourishment is refused, artificial feeding must be resorted to. Stimulants are usually required in severe cases. The bowels must be kept open, and salines are recommended for this purpose. For violence the patient must be placed upon a mattress on the floor and surrounded by other mattresses. For persistent insomnia hypnotics must be used. Sulphonal, 30 grains for a child over 13 years, for adults this can be repeated morning and evening if necessary. In agitated cases morphia bimeconate is often useful. Chloral is also recommended.

**Choreomania.**—The dancing mania, epidemic in character, chorea Germanorum.

**Chorioma.**—Chorion-epithelioma or syncytioma malignum. See PREGNANCY, OVUM AND DECIDUA (*Epithelioma of the Chorion*), PUERPERIUM, PATHOLOGY (*Sarcoma-Deciduo-Cellulare or Deciduoma Malignum*).

**Chorion.**—The outer foetal membrane. See FÆTUS AND OVUM, DEVELOPMENT (*Fœtal Membranes, Placenta*), LABOUR, RETENTION OF PLACENTA (*Fragments of Membrane*), PHYSIOLOGY, REPRODUCTION (*Development*), PREGNANCY, DIAGNOSIS (*Hydatid Degeneration of the Chorion*), PREGNANCY, AFFECTIONS OF THE OVUM AND DECIDUA (*Vesicular or Hydatid Mole*), PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Causes of Death during Pregnancy*), PREGNANCY, HÆMORRHAGE (*Antepartum, Myxoma Chorion*).

**Chorion-Epithelioma.** See PREGNANCY, OVUM AND DECIDUA (*Epithelioma of the Chorion*), PUERPERIUM, PATHOLOGY (*Sarcoma-Deciduo-Cellulare or Deciduoma Malignum*).

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See also ACCOMMODATION (*Changes in the Choroid*), CHERK, FISSURE OF, EYEBALL, INJURIES OF (*Rupture of the Choroid*), MENINGITIS, TUBERCULOUS AND POSTERIOR BASIC (*Ophthalmoscopic Examination*), OCULAR MUSCLES, AFFECTIONS OF (*Nystagmus, Causes*), PALATE (*Facial Cleft, Coloboma of Choroid*), PHYSIOLOGY, THE SENSES (*Vision*), PIGMENTS OF THE BODY (*Choroid*).

The choroid is the posterior part of the *tunica vasculosa*, the anterior part being formed by the iris and ciliary body. In its extent it reaches from the ciliary body to the optic disc, and lies between the sclerotic externally and the retina internally. On microscopic section it presents, from without inwards, the following layers:—

1 *The lamina fusca.*—This consists of lamellæ of loose connective tissue, containing branching pigment cells, it adheres to the sclerotic when that is separated from the choroid, and therefore it is sometimes described as belonging to that tissue.

2 *The lamina suprachoroidæ* is similar in structure to the lamina fusca, being composed of lamellæ of branched pigmented connective-tissue corpuscles and a network of elastic tissue. The space between this layer and the last is lined by endothelium, and is considered to be a lymph space.

3 *The lamina vasculosa* consists of a dense network of large intercommunicating veins so closely connected that in some parts the intervessel spaces are less in diameter than the veins themselves. This venous plexus ends abruptly at the ora serrata. These veins are the tributaries of the vena vorticosæ. Within the fenestrations, which are chiefly fusiform in shape, are pigmented connective-tissue corpuscles, the presence of which renders this membrane uniformly dense and reflective in most eyes, a deficiency or excess of these pigment-cells will cause the outline of the choroidal vessels to be seen.

4 *The chorio-capillaris, or membrane of Ruysch*, is a reticulated vascular membrane of closely intercommunicating capillaries of large diameter, the meshes of this network are small. Here the vena vorticosæ begin in capillary whorls, the *stars of Winslow*. There are no pigment-cells in this layer, and few, if any, round cells. A delicate structureless membrane, the *elastic layer of Sattler*, is supposed to exist between this and the former layer. This Sattler believes to be the remains of the *tapetum*, a definite layer found in the choroid of certain animals. In carnivora the tapetum is composed of endothelial cells containing minute crystals, producing a shining appearance in the dark. In other ani-

mals it is fibrous in nature, and, since the retinal pigment is absent from certain spots, interference of light is produced, causing a similar appearance.

5. *The lamina vitrea, or membrane of Bruch,* is an hyaline membrane. It is continued forwards into the ciliary body and iris. It supports the pigmentary epithelium of the retina, and just as this layer, formerly thought to belong to the choroid, has been shown to be epiblastic in origin, so the lamina vitrea is in all probability a layer of the retina, and not of the choroid, being produced originally from the cells of the pigment layer of the retina.

The choroid is almost completely supplied by the *short posterior ciliary arteries*. They are ten or twelve in number, and pierce the sclerotic close to the optic nerve, passing through the lamina fusca into the deeper part of the lamina suprachorioidea, they divide dichotomously, and pass into the capillaries of the chorio-capillaris. Except in the region of the optic nerve, where a circular arterial anastomosis exists around the disc with small branches of the *arteria centralis retinae*, the anastomosing branches being called the *cilio-retinal arteries*, the branches anastomose little with each other. Anteriorly they receive a few communications from the arteries of the ciliary region.

The *veins* of the choroid anastomose very freely together, they do not accompany the short posterior ciliary arteries, but are arranged in curves, *venae vorticosae*, as they converge to four or five principal trunks, these pierce the sclerotic very obliquely about half-way between the optic nerve and cornea to join the ophthalmic vein.

The *lymphatics* of the choroid help to regulate the intra-ocular pressure. Between the lamina fusca and the lamina suprachorioidea is a lymph space, which communicates by means of perivascular sheaths surrounding the *venae vorticosae* with the lymph space within the capsule of Tenon.

The *nerves* of the choroid are derived from the long ciliary branches of the nasal branch of the first division of the fifth nerve, and from the short ciliary branches of the lenticular ganglion. They are chiefly vaso-motor in action, and form in the lamina suprachorioidea a plexus, in the meshes of which ganglion cells are found.

**THE APPEARANCE OF THE HEALTHY CHOROID** must be carefully studied before we can diagnose pathological conditions.

The colour of the *fundus oculi* seen by reflecting light from the ophthalmoscope is due to the following causes — (1) The blood contained in the chorio-capillaris and lamina vasculosa, (2) the pigment-granules contained in the pigmented layer of the retina, the interstices of the vascular layer of the choroid, and the lamina fusca and lamina suprachorioidea, and (3) the sclerotic, which reflects a certain amount of light through the retina and choroid.

When pigment is altogether absent from both retina and choroid, as in *albinos*, we get a light yellowish red colour reflected from the blood within the capillaries, whilst the interstices between the latter are seen to be of a lighter, almost white appearance, owing to the reflection from the sclerotic beyond the lamina fusca, and thus a fairly well-defined outline of the choroidal vessels is obtained.

In *fair persons*, where the pigment-granules contained within the cells are of a faintly brown colour, the fundus is yellowish red, and the vessels of the choroid can often be seen, although less distinctly than in albinos.

In *moderately dark persons* this pigment becomes of a deeper brown, and all details of the choroid are hidden, the fundus presenting a light brownish red colour, with no visible choroidal vessels. If, however, as is occasionally the case, pigment is scarce in the pigmented layer of the retina, while abundant in the choroid, a network of red vessels is seen upon a dark background, the so-called *choroide tigrée*.

In *negroes*, and all *dark races*, the pigment is so abundant as to prevent almost all red choroidal reflex, the fundus assuming a brownish grey, or even a slate colour.

The colour of the fundus varies very much with the intensity and colour of the light used, with the state of dilatation of the pupil, and with the refractive condition of the eye. It is brighter, *ceteris paribus*, in proportion to the number of rays of light that can be thrown into the eye.

**CONGENITAL AFFECTIONS OF THE CHOROID** — *Coloboma of the choroid* is a congenital deformity, consisting in the absence of a more or less considerable portion of this part of the tunica vasculosa, and is usually found in the lower and internal part of the globe. When examined by the ophthalmoscope it appears of a bright bluish white colour, with clear cut pigmented edges. The surface of the sclerotic often appears very irregular, is crossed by retinal vessels, and frequently has upon it small aggregations of pigment. The extent of the coloboma is very variable, it usually reaches from the edge of the optic disc nearly as far as the ciliary body. It may embrace the optic disc, in which case the latter is changed in appearance, and looks as if it were hyperæmic by contrast with the white area round it. It may occur in the yellow spot region, *macular coloboma*, it may be very localised, normal choroid being seen all round it, it is often accompanied by coloboma iridis. The retina is often involved in the coloboma, though it may be present over the whole site. Occasionally coloboma of the choroid occurs in both eyes, but when unilateral the left eye is most commonly affected. There is a large scotoma in the visual field corresponding to the coloboma, but otherwise the sight in most cases is fairly good. The scotoma

is not necessarily absolute, as the retina may be present.

The cause of the defect is probably due to the formation of adhesions between the developing retina and the mesoblast, the latter of which is not differentiated into choroid and sclerotic. This usually takes place in the position of the retinal fissure, and thus accounts for the usual position of the coloboma. It may, however, take place in any part of the retina, hence the occasional occurrence of the coloboma elsewhere.

*Albinism*, or congenital absence of pigment throughout the body, may be relative or absolute. In *absolute* albinism the pupils and irides appear pink from the reflected light from the choroid, the irides also on account of the blood contained in their vessels. This condition is usually attended with defective visual acuity, photophobia, and nystagmus. Upon ophthalmoscopic examination, the choroidal vessels are seen most distinctly as a pink fenestrated membrane upon a pale, almost white, background. The hair is usually white throughout the body. In *relative* albinism, the hair is a pale straw colour, the irides present a pale purplish colour, and do not completely shut off the choroidal reflex, the symptoms also are less marked. There is a tendency to acquire pigment as the child grows, the improvement, however, is rarely more than a change from the absolute to the relative condition. By way of treatment, any existing error of refraction must be corrected, dark glasses should be worn, with or without a diaphragm, to cut off some of the light. Peripheral tattooing of the cornea has been performed for the same object.

*Congenital crescent* is a peculiar greyish white crescentic patch immediately below or, in rarer instances, to the outer side of the optic disc. The disc, excluding the crescent, is usually oval, but with it appears circular or slightly oval in the opposite direction. These crescents present no marginal pigment, which is so often seen in myopic crescents, besides, the latter are usually to the outer side of the disc. It is very probable that this crescent is a partial coloboma of the choroid.

*Choroiditis*.—Inflammation of the choroid is accompanied by infiltration and exudation, which may be reabsorbed with or without atrophic changes, or may pass into the stage of suppuration. If the morbid process be limited to the choroid proper, it is frequently unattended by external signs of inflammation, being recognised chiefly by visual troubles and ophthalmoscopic appearances. When the other portions of the uveal tract—the ciliary body and iris—are involved in the process, as is usually the case in acute and suppurative forms, the external signs of these inflammations are always present. The two chief varieties of choroiditis are plastic or exudative choroiditis and suppurative choroiditis. Besides these, we have forms of choroiditis accompanied by inflammation of

neighbouring parts, these are—irido-choroiditis, retino-choroiditis, and sclerotic-choroiditis.

*PLASTIC OR EXUDATIVE CHOROIDITIS—Etiology*.—The most common cause is syphilis, inherited or acquired. In the acquired form it usually occurs towards the end of the first year after the primary infection, or during the beginning of the second year. Inherited syphilitic choroiditis is commonest between the ages of six months and three years, it may occur, however, much later together with interstitial keratitis. Other causes of plastic choroiditis are tuberculosis, gonorrhoea, the simple and profound anemias, and menstrual disorders. Many chronic cases occur to which no definite cause can be assigned.

*Symptoms*.—Plastic choroiditis is the most common form, and presents itself under several forms, the symptoms varying with the extent of the area affected, and with its position with regard to the macular region. When unattended with cyclitis or iritis there are no external signs of the disease. *Subjective symptoms* are usually the earlier and more important indications of the disease, but they may be completely absent although the ophthalmoscope shows gross changes. The patient complains of seeing lights of a pale blue or red colour—*phosphenes*—at night, when the eyes are closed and the room is dark. In the daylight he sees large floating specks, especially when looking at a white object, these are larger than the ordinary muscae volitantes, and tend to obscure the vision by settling upon one portion of the object looked at. Distortion of the outline of objects—*metamorphosis*—is also a characteristic feature, especially when the exudative change is situated in the macular region, if parallel straight lines are held before the eye they will appear curved in various ways. *Micropsia*—objects appearing smaller than normal—and *macropsia*—objects appearing too large—are symptoms sometimes present. The patient may complain of dark spots in the visual field—*positive scotomata*,—or these may only be found by carefully testing the field with the perimeter—*negative scotomata*. It will then be found that its whole area is not intact, but that, according to the position and extent of the disease, there will be small and large areas in which vision is either defective or altogether absent—*relative or absolute scotomata*. The light sense is also appreciably diminished, especially when there is cloudiness of the vitreous. In some cases the patient will complain of a dull aching pain at the back of the eyes.

*Ophthalmoscopic Signs*.—Recent patches of choroiditis appear as yellowish, ill-defined areas, lying beneath the retinal vessels upon and in the red background of the choroid. These patches may be more or less obscured by a hemorrhage from a choroidal vessel, this being sometimes the earliest sign of the disease. The yellow exudation may entirely disappear after

some weeks, leaving, in rare cases and under proper treatment, the choroid intact, but more commonly, as it disappears, the affected area is found to be more or less atrophic, gradually becoming whiter, with the choroidal vessels showing up, pigment at the same time appearing round each patch and within some of them, crystals of cholesterol may be seen with some of the patches. Still later the choroidal atrophy presents the appearance of white patches, ringed by pigment, with no traces of choroidal vessels, but crossed uninterruptedly by the retinal vessels. It is not uncommon to find both old and recent patches in the same eye. The inflammation usually attacks the adjacent retina, indeed, the exudation often extends through the retina into the vitreous. The vitreous is, therefore, often cloudy owing to the presence of fine opacities. This is more particularly the case in syphilitic chorio-retinitis. The nebulous condition of the vitreous not only interferes with the patient's vision, but prevents the details of the fundus from being clearly seen, the existence of cloudiness of the vitreous is, therefore, always suggestive of choroiditis, although hyalitis without choroiditis is sometimes found. Care must be taken to exclude a nebulous cornea, as this will cause a haziness of the fundus very similar to that produced by fine vitreous opacities. On the other hand, a hazy vitreous will make the outline of the optic disc appear blurred, and thus simulate papillitis.

**Pathology**—When a recent patch is examined microscopically, we find a cluster of round and fusiform cells in the lamina vitrea and the chorio-capillaris, with great engorgement of the vessels, and perhaps one or two hemorrhages. The pigment-layer of the retina is then unaffected. As the disease progresses the cells of the pigment-layer proliferate, and the part which is immediately opposite to the patch becomes absorbed, giving it a white appearance, the pigment becomes accumulated at the edges of the patch, and the inflammation extends to the layer of rods and cones, and the outer granular layer of the retina, with small-celled infiltration. Later, this inflammatory exudation becomes absorbed, and gives place to cicatricial tissue, but the structures involved—viz the outer granular layer, the rods and cones, the uveal tract, and the vessels of the choroid—are destroyed, and their place occupied by this new connective tissue.

**Varieties**—Many different kinds of plastic choroiditis are found. The following may be mentioned as those more frequently met with: (1) Disseminated, (2) Central, (3) Peripheral, (4) Diffuse exudative, (5) Myopic.

(1) *Disseminated choroiditis* consists of numerous round or irregular spots scattered over the fundus. These pass through the changes mentioned above, and mostly end in patches of complete atrophy. They may become confluent,

involving the whole or the greater part of the fundus. The optic nerve may become involved in the process, in which case it becomes first hyperæmic and finally atrophic. This form of choroiditis may easily be mistaken for *guttate choroiditis of Tey* (p. 145).

(2) *Central choroiditis* consists of exudation in the macular region, forming an irregular, more or less circular patch, with considerable pigmentation. Vision is impaired, and a large central scotoma is present. This is especially frequent in old people, where a very chronic inflammation, without much pigmentation or exudation, is followed by well-defined atrophic changes. In such cases the fenestrated arrangement of the choroidal vessels is clearly seen. This senile form is usually bilateral, and must be carefully sought for before the extraction of all senile cataracts, as its presence will greatly modify the prognosis. Before a diagnosis of amblyopia is made, either congenital or toxic, the macular region should be carefully examined for central choroiditis.

(3) *Peripheral or anterior choroiditis* is that form in which only the peripheral parts of the choroid are affected. Owing to the outlying position of the lesion, direct vision is but little disturbed, and the disease may be easily overlooked unless the peripheral portions of the fundus are carefully examined.

(4) *Diffuse exudative choroiditis*, extending over the whole fundus, is sometimes associated with syphilis. Although the whole choroid is affected, yet the deposit of lymph appears to assume the form of circumscribed patches, varying somewhat in tint from a yellowish red to white, with pigmentation according to the stage of the affection. It is always more or less obscured from view by fine dust-like opacity of the vitreous, and not infrequently large membranous floating opacities of the vitreous are present. The vitreous and the retina may ultimately become clear, and then the ophthalmoscope reveals more definitely the large atrophic patches in which masses of pigment are here and there distributed. Such cases, when they have arrived at an advanced stage, are often difficult to distinguish from *retinitis pigmentosa* (see "Retinitis"), for the retinitis secondary to syphilitic choroiditis tends to simulate that disease. Peripheral retinal pigmentation, night-blindness, and contraction of the visual field occur, and it is only by the presence of vitreous opacities, patches of choroidal atrophy, relative distension of the veins, together with perhaps concurrent nitis or cyclitis, and a definite history of syphilis, that the right diagnosis can be arrived at. The visual field, too, may help, for in many instances there is no peripheral contraction, but a ring scotoma, which is almost pathognomonic of syphilitic chorio-retinitis.

This kind of choroiditis almost invariably affects the retina to a considerable extent

(5) *Myopic choroiditis*, see *Sclerotic-choroiditis*

**Prognosis of Plastic Choroiditis**—Plastic choroiditis runs a chronic course, its worst feature is a tendency to recurrence. Many cases are so chronic that they extend over years without perceptible change, while others present new spots of exudation from time to time, until the choroid is ultimately covered with atrophic patches. In bad cases the optic nerve and retina are involved, and partial or total blindness results. Vision, however, may be almost completely restored so long as the macular region is free. Central changes, like peripheral, are attended with subsequent atrophy, which gives rise to permanent and absolute scotomata; consequently, direct vision becomes completely lost. Diffuse exudative choroiditis is invariably attended with gradual failure of sight until complete blindness ensues.

**Treatment of Plastic Choroiditis**—It is necessary to find out, if possible, the cause of the disease. A history of syphilis can often be elicited, and should the disease be active, mercury in some form must be administered, either with or without iodide of potassium, the subconjunctival injection of mercury has been warmly advocated by various Continental surgeons, and the discussion of this and other methods of treatment will be found under the head of "Iritis." Other constitutional causes require general treatment. Locally, if there is active inflammation, as shown by dull pain, diffuse vitaceous haze, and fresh exudations, leeches applied to the temple, diaphoresis by hot-air baths or pilocarpine hypodermically injected, and profuse purges, are necessary. Counter-irritation by means of the Argyll-Robertson method frequently helps to clear up the condition. This consists in rubbing into the skin of both eyelids pure silver nitrate until vesication is produced. The eyes should be protected from the light by tinted glasses, they should be rested as much as possible, and no near work attempted. It may be necessary to atropinise the eyes to prevent accommodation.

Should there be no history of syphilis, it is in many cases still advisable to try the mercurials and iodides, as great improvement is often obtained by their use independent of any syphilitic history. If the general health is run down it must be supported by a nutritious diet, regular outdoor exercise, and by the exhibition of tonics, such as iron, strychnine, quinine, etc., local treatment must also be adopted as indicated above. It is advisable in most cases of choroiditis to avoid alcoholic stimulants.

**SUPPURATIVE CHOROIDITIS** is a diffuse suppurative inflammation of the choroid, and is always attended with suppurative irido-cyclitis. There is, in fact, a general inflammation of the tunica vasculosa, which usually spreads to the other

tunics of the globe, and gives rise to that condition known as *panophthalmitis*.

**Etiology**—The causes of suppurative choroiditis may be divided into three groups—

(a) Injury, of a penetrating nature, if septic infection by pyogenic matter is produced. Operative measures on the eyeball, where due aseptic precautions have not been taken, may be placed under this category.

(b) There may be an extension of an inflammation elsewhere, such as from a septic ulcer of the cornea.

(c) Inflammation of the choroid may be the result of metastasis. Of metastatic choroiditis the most important is the puerperal form, occurring as a symptom of pyæmia, usually in the second week after delivery. It is also occasionally found in ulcerative endocarditis, cerebrospinal meningitis, and during the acute specific fevers. It is due to septic embolism of the retinal and choroidal vessels.

The *symptoms* from the first are those of intense inflammation. The eyelids are red, swollen, and oedematous, so that it is with difficulty that the eye can be examined, were it not for the absence of discharge, the condition might be mistaken for purulent conjunctivitis. There is great chemosis and conjunctival injection. The cornea is hazy and anæsthetic, the iris muddy and immovable, with a somewhat dilated pupil. The anterior chamber is shallow and often contains pus (*hypopyon*). There is slight proptosis, and the tension is somewhat raised. If the media are sufficiently clear the exudation into the vitreous can be seen as a yellowish reflex.

As time goes on there may be a gradual subsidence of the symptoms, the proptosis diminishes, the inflammation subsides, the tension falls, and gradually a general shrinking of the whole globe (*phthisis bulbi*) occurs. In the acute form, however, suppuration occurs, with marked constitutional symptoms. High fever, vomiting, and violent pains ensue, the proptosis increases, the lids become more swollen, until the eyeball gives way anteriorly, with escape of pus and disappearance of the acute symptoms.

**Pathology**—Suppurative irido-choroiditis is attended with suppurative retinitis, the vitreous also, participating in the progressive and destructive inflammation (suppurative hyalitis), becomes destroyed and replaced by pus, and the eyeball is converted into an abscess-cavity. In such a state either the cornea will slough and the pus be discharged, or the pus will become inspissated by absorption of its liquor puris, in either case the globe will shrink. In many cases the exudation in the vitreous is more puriform than purulent, and the yellowish white reflex seen has a somewhat similar appearance to gloma retina. Hence it has been termed *pseudo-gloma*. The puriform exudation often undergoes partial organisation, and so the

ophthalmoscopic condition may remain for some weeks after the subsidence of active inflammatory changes, eventually, however, the eyeball is sure to shrink, become puckered and hardened, and in some cases the choroid will undergo ossification.

Microscopically the choroid will be found to be greatly thickened and exceedingly cellular, the cells aggregating into clusters which form small abscesses, these soon coalesce and convert the choroid into a diffuse suppurative tract. The inflammatory changes in the iris, ciliary body, retina, and vitreous are similar to the microscopical characters of inflammation found in other parts of the body. It is advisable to mention, however, that the partially organised exudation in the ciliary body looks under the microscope not unlike a melanotic sarcoma, on account of the complete derangement and multiplication of the pigment-cells. The clinical history, together with the condition of the vessel-walls, will explain, however, its nature.

In metastatic choroiditis it is often possible to find the infecting emboli, which can be shown to contain pyogenic organisms, the streptococci being the most common.

**Diagnosis.**—The main difficulty is to distinguish between a chronic and subacute form of suppurative choroiditis and certain intra-ocular growths, of which glioma is much the most common. The diagnosis depends chiefly on the history, the presence or absence of recent inflammatory signs, and the tension of the globe. Suppurative choroiditis is usually preceded by a perforating injury, or by a severe illness such as a specific fever. Again, signs of recent iritis will be seen, and, lastly, intra-ocular neoplasms, though at first they may be present in a globe of normal tension, soon produce a rise of tension with all the signs of secondary glaucoma—suppurative choroiditis, on the other hand, causing a fall in the tension. Other points to help will be the age of the patient, glioma only occurring in young children, and the appearance of the reflex, which is of a much more glistening appearance in glioma than in pseudo-glioma.

**Prognosis.**—The diseased eye is sure to be lost, so far as sight is concerned, and will inevitably pass into a state of atrophy. In all cases the question of sympathetic inflammation in the other eye has to be carefully considered (see "Sympathetic Ophthalmitis").

**Treatment.**—Local leeching, hot fomentations, combined with morphia, either hypodermically or by the mouth, are useful in allaying pain. As soon as pus is evidently accumulating in or behind the aqueous chamber prompt surgical interference is indicated. If the eye be left to itself there is considerable risk of the inflammation extending along the optic nerve to the brain and its membranes, and so causing a fatal termination. Excision of the globe is, in my

opinion, the best and safest way of treating this severe condition, great pains being taken after the enucleation to treat the socket antiseptically. Some surgeons, however, are doubtful as to the propriety of removing an eye whilst in this inflamed and suppurating condition, and prefer first to make an incision through the anterior part of the globe so as to relieve pain, tension, etc., and to give a free outlet to the pus, postponing the excision until the inflammatory symptoms have subsided.

**IRIDO-CHOROIDITIS** (uveitis) is an inflammatory condition involving the whole uveal tract. It may be *acute* or *chronic*.

**Acute Irido-Choroiditis** is either suppurative (see "Suppurative Choroiditis") or sero-plastic (see "Sympathetic Ophthalmitis").

**Chronic Irido-choroiditis** attacks each part of the uveal tract either simultaneously or successively. It is very often preceded by an acute attack of *iritis* or *choroiditis*. Subacute exacerbations occur from time to time. Its tendency to relapse is its predominating feature, years may pass away before there is a permanent cessation of inflammatory symptoms. The prevailing clinical features are evidences of *iritis*, *cyclitis*, and *choroiditis*, such as posterior synechiae or pigment on the lens capsule, thinning of the nitic tissue, so that in some cases the dull choroidal reflex may be seen through the reticulum of the iris, as if through a curtain, thinning of the sciera over the ciliary body, from chronic *cyclitis*, which allows the blue ciliary body to be seen through, eventually a ciliary staphyloma may appear. Numerous and large vitreous opacities are seen, and, if the vitreous is not too cloudy, patches of *choroiditis* and *choroidal atrophy*, with general thinning of the choroid, may be made out. Posterior polar cataract or irregular opacities in the lens are not uncommon, and at last the whole lens may become cataractous. Subjective signs are present, namely, defective vision, sometimes amounting to bare perception of light, phosphenes, dull aching pains in the eyes, etc. The tension is usually slightly raised at first, but soon becomes subnormal (T-1 or T-2). The prognosis is unfavourable. The disease is usually symmetrical, and complete blindness will almost certainly ensue sooner or later.

**Treatment.**—Little can be done beyond treating the symptoms and supporting the general health. Iodide of potassium assists in some cases, chiefly on account of its alterative action and its powers in aiding the absorption of all chronic inflammatory exudations. Mercurial inunctions are also useful for the same purpose. Iritis must be treated in the early stage by instilling atropine. Increase of tension is rarely present, and certainly is never sufficiently pronounced to necessitate operative interference.

**RETINO-CHOROIDITIS.**—There is almost always some accompanying retinitis with all cases of

plastic choroiditis. It is unnecessary to add anything to what was said under that heading.

**SCLEBOTIC-CHOROIDITIS, OR MYOPIC CHOROIDITIS.**—This is rather of the nature of an atrophic condition of choroid than a true inflammation, and will be referred to under "Myopia."

**TUBERCULOUS DISEASE** may affect the choroid in three ways, which differ in ophthalmoscopic signs, prognosis, and treatment.

(a) *Tuberculous Disseminated Choroiditis*.—This form of chronic inflammation of the choroid is very similar to the syphilitic variety, and is, according to some authorities, as common. It is with difficulty distinguished, but the patches are smaller and lightly raised. Just as in the syphilitic form, degeneration takes place with choroidal atrophy, the process being, however, slower. Its treatment is unsatisfactory; before atrophic changes have set in mercurialunction may help absorption of the exudation, and phosphates or arsenates with cod-liver oil should be prescribed by the month. The prognosis is not so good as in the syphilitic variety.

(b) *Miliary Tuberculosis of the Choroid* occupies the region of the chorio-capillaris and the vascular layer, and is quite behind the uvea. It is most commonly found in cases of acute miliary tuberculosis, but it may be present in all forms and stages of tuberculous disease.

Ophthalmoscopically it appears as a greyish hemispherical eminence varying from 3 mm to 1 mm in diameter, and may be even smaller. One or several of these first appear in the macular region, and are afterwards followed by others in the neighbourhood. The youngest tubercles are very small, the oldest are the largest, and are somewhat white at the centre. The ophthalmoscopic signs are very similar to those of disseminated choroiditis, though the two conditions are not likely to be mistaken owing to the different general conditions. The patches are more raised, rarely pigmented, more clearly defined, and less brilliantly white. In the form that occurs in acute general tuberculosis they appear as a rule only a short time before death. Papillitis is a frequent accompanying symptom. Both eyes are nearly always affected.

Microscopically each patch shows the typical structure of tubercle, though the specific bacilli are not always to be found.

In cases of acute tuberculous disease where there are typhoid symptoms, and in tuberculous meningitis where the diagnosis is not always easy, the detection of tubercles of the choroid is of great assistance in clearing up the case, although the absence of choroidal tubercle does not prove the absence of tuberculous disease in other organs.

(c) *Tuberculous Tumour of the Choroid*.—This condition is probably always secondary to tuberculous disease elsewhere, though this cannot

always be proved. In early stages, a retinal detachment, less defined than the detachment due to choroidal sarcoma, will be the only sign. As growth takes place glaucomatous symptoms rarely occur, the coats of the globe rapidly giving way with the formation of a staphyloma and subsequent panophthalmitis. It is doubtful if general infection can occur, so enucleation should be postponed till vision is lost. The growth is always unilateral.

**GUMMA OF THE CHOROID** is very rare. It may exist in conjunction with gumma of the iris or ciliary body. The diagnosis is necessarily difficult, inasmuch as the vitreous is hazy and the fundus cannot be seen. It rests mainly on the concurrent syphilitic lesions, the history, and the effect of antisyphilitic treatment. Sight is usually greatly impaired, but, if the condition is attacked in time, almost perfect vision may be regained.

**SARCOMA OF THE CHOROID** is the commonest intra-ocular tumour of adult life, just as gloma is the most frequent in infancy and early childhood.

**Etiology**.—No cause has been discovered. There appears to be no relationship between it and blows on the eyeball.

**Symptoms**.—Sarcoma of the choroid usually begins in a manner so insidious as to be unnoticed even by the patient until the tumour has attained a considerable size, even then it is often discovered accidentally. Sometimes, however, though rarely, the growth of the sarcoma is accompanied by local pains, phosphenes, etc.

When seen at an *early stage* there may be nothing externally to attract notice. In addition to the dimness of sight which may have first caused the patient to apply for advice, we find that the visual field is defective, and, when it is examined by means of the perimeter, presents a scotoma corresponding to the position of the tumour. With the ophthalmoscope the outline of the tumour can sometimes be seen to form a rounded prominence, pushing the retina forwards into the vitreous cavity. In this early stage inflammatory signs are absent, and the tension is normal or even slightly subnormal.

At a *later stage* the presence of the tumour is accompanied by a distinct increase in the tension of the globe, and the eye presents other symptoms of glaucoma. The anterior ciliary vessels are congested, the cornea becomes hazy and more or less anæsthetic. The anterior chamber gradually becomes shallow. The iris is sometimes subacutely inflamed, and forms posterior adhesions (*synechia*) to the capsule of the lens, which render the pupil irregular. Not infrequently the iris is atrophied, and it may be detached at that part of its periphery which corresponds to the position of the tumour. The vitreous also is frequently rendered cloudy by the presence of opacities. The vision has gradu-



ally become worse, and is now reduced to bare perception of light. When the disease has progressed so as to destroy vision there is frequently considerable pain in the ciliary region and lachrymation, more severe than in true glaucoma.

In the *third stage* the tension of the globe is suddenly reduced, signifying that the sclerotic has given way, and the glaucomatous symptoms are relieved. The growth, however, now rapidly extends and infiltrates all the surrounding structures.

**Pathology.**—Sarcoma of the choroid may be divided into the following varieties:—

*A* Leuco-sarcoma—(a) Spindle-celled, (b) round-celled, (c) mixed-celled.

*B* Melano-sarcoma—(a) spindle-celled, (b) round-celled, (c) mixed-celled.

*C* Intervening grades of pigmentation.

Of these, spindle-celled melano-sarcoma is the commonest. Leuco-sarcoma is found in about one case in every eight or nine, but even then a few pigment-containing cells are present. When the growth is white the cells are usually of the round variety, and it appears probable that this form starts from the non-pigmented chorio-capillary layer, while the melanotic form has its origin in the deeper pigmented layers.

The tumours usually are firm, they generally contain some blood-vessels, and sometimes are very vascular, the walls of the blood-vessels are composed of sarcomatous elements. They have an even and smooth convex surface so long as the lamina vitrea remains intact. When they perforate this membrane they grow more rapidly, become hour-glass in shape, and present an irregular granular surface. Sometimes there is also effusion of serum or blood beneath the retina. They may increase so as to fill the whole globe and distend its walls before invading the extra-ocular tissues, but in many cases the tissues outside the sclerotic are affected by the new growth, whilst the tumour within the globe is quite small, in these cases the cells pass to the outside by means of the sheaths of the blood-vessels, which are seen to be thickened and altered by the presence of cells similar in character to those of the tumour.

The neighbouring lymphatic glands are not affected, but secondary sarcoma is liable to be set up elsewhere by a process of embolism, the cells being conducted from the primary source by the blood-current. The liver is the organ usually first affected.

**Diagnosis.**—It is obvious how important an early diagnosis is, but this is often extremely difficult. In the early stage there is always great doubt, whether the ophthalmoscopic appearance of the retina is due to a simple detachment from subretinal effusion or to a sarcomatous growth in the choroid. When the latter is the case the detached retina may retain some colour or be pigmented, it may occur

at any part of the fundus, and it does not flap about when the eye is moved. In simple detachment the detached portion is bluish white, usually occurs at the lower segment of the fundus, and may flap about freely when the eye is moved. Occasionally a vascular network of the sarcomatous growth can be detected through the retina. In any case of extensive detachment occurring in one eye only, and when there has been no myopia or history of injury, we must suspect sarcoma of the choroid.

In the stage of increased tension sarcoma of the choroid may easily be mistaken for acute primary glaucoma. If the fundus can be seen there is usually no difficulty, but if this is impossible, the diagnosis must be made from the history of the case, whether any symptoms of glaucoma had previously existed, and by the condition of the visual field and the projection of light. An important point in the history is the relation between the onset of pain and the loss of acuity of vision. In primary glaucoma the two are almost synchronous, whereas in choroidal sarcoma the sight in most cases has been gradually getting worse previous to the glaucomatous symptoms.

**Course and Prognosis.**—If left alone the disease usually takes some years to run through all its stages, the end being always fatal, the patient dying generally from extension into the brain or from metastasis. In all cases, however early the diagnosis is made, the eye is lost, and we can never promise that there will be no recurrence, local or general.

**Treatment.**—Enucleation should be performed as early as possible, together with excision of 4 or 5 mm. of the optic nerve. If the growth has extended outside the globe the orbit should be emptied.

**CARCINOMA OF THE CHOROID** is very rare, and is always secondary, usually to carcinoma of the breast. Death usually occurs within twelve months.

**CHOROIDAL DEGENERATION.**—*Guttate choroiditis of Tay* is characterised by a number of yellowish-white specks usually, though not necessarily, in the macular region. They are probably spots of colloidal degeneration of the choroid.

**Ossification of the choroid** frequently follows phthisis bulbi. It may be extensive, a thin plate being present on the inner or vitreous part of the choroid, or there may be merely a few spicules of bone scattered throughout the choroid.

**INJURY TO THE CHOROID.**—Rupture of the choroid is always the result of external violence, such as a blow or a fall. The accident is usually followed by hemorrhage into the vicinity of the wound, causing opacity of the vitreous. After a few days, as the blood becomes absorbed, we can see a whitish line in the fundus, with a little blood clinging to its edges. It is usually curved, concentric with the edge of the disc.

Subsequent to the rupture there is frequently a tendency for masses of pigment to appear round the exposed sclera.

**Concussion Choroiditis**.—Rupture of the choroid is often accompanied by macular choroidal degeneration

**Choroideremia**.—Congenital absence of choroid or of its epithelium

**Choroiditis**. See CHOROID, DISEASES OF (*Choroiditis*), see also AMBLYOPIA (*Symptoms*), COLOUR VISION (*Acquired, Choroido-retinitis*), CORNEA (*Interstitial Keratitis*), MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Symptoms, Special Senses, Eyes*), SYPHILIS (*Tertiary, Eye, Choroiditis*), SYPHILIS (*Children, Eye, Choroiditis*), VISION, FIELD OF (*Central Choroiditis*)

**Choroido-cyclitis**.—Inflammation affecting both the choroid and the ciliary body See CHOROID, DISEASES OF (*Irido-choroiditis*), IRIS AND CILIARY BODY (*Cyclitis*), GLAUCOMA

**Choroido-iritis**.—Inflammation of both choroid and iris See IRIS AND CILIARY BODY (*Iritis*).

**Choroido-retinitis**.—Inflammation of the choroid, extending to the retina See CHOROID, DISEASES OF (*Retino-Choroiditis*)

**Choroid Plexus**. See BRAIN, PHYSIOLOGY (*Lymphatic Circulation*), HYDROCEPHALUS (*Etiology*), PHYSIOLOGY, NERVOUS SYSTEM (*Brain*)

**Chrom-**.—In compound words *chrom-*, or *chroma-*, or *chrome-* means "relating to colour," as in *chromatolopsis* (colour-blindness), *chromatophobia* (sensitiveness of the eye to certain colours), *chromatosis* (a morbid condition of the skin as to pigmentation), *chromocyte* (a red blood corpuscle), etc., as well as in the words following

**Chromatin**.—The fibres of the cell nucleus which are stained by various dyes, and which contain much nucleic acid, are composed of a substance called chromatin See PHYSIOLOGY, THE CELL (*Nucleus*)

**Chromatolysis**.—Destruction of the chromatin material in cells so that their staining power is lost (*achromatous*), this change occurs in a nerve cell after the division of its axon, and is usually temporary See PHYSIOLOGY, NERVOUS SYSTEM (*Spinal Cord*); INSANITY, PATHOLOGY (*Cortical Nerve Cells*).

**Chromatopsia**.—That condition in which all objects seem to be of a certain colour (e.g. grey), partial colour-blindness See HYSTERIA (*Sensory Disorders, Ocular Anesthesia*)

**Chromaturia**.—The state of abnormal coloration of the urine

**"Chrome Holes."**—The cutaneous ulcers occurring in workers in the bichromate industry See TRADES, DANGEROUS (*Workers in the Chemical Trades*)

**Chromidrosis**.—The secretion of coloured perspiration, especially on the face and eyelids See SKIN, DISEASES OF SWEAT AND SEBACEOUS GLANDS (*Chromidrosis*), SKIN, PIGMENTARY AFFECTIONS OF (*Spurious Pigmentations*), also EYELIDS, AFFECTIONS OF (*Skin of Lids, Chromidrosis*)

**Chromism**.—Chronic chromium poisoning occurring in workers in chemicals, and characterised by ulceration of the nasal septum and by "chrome holes" (qv) on the skin See also TOXICOLOGY (*Irritants, Chromium*)

**Chromium**.—There are two forms in which chromium occurs in the Pharmacopoeia *Chromic acid*,  $\text{CrO}_3$ , readily soluble in water, having a powerfully caustic action, and being used as a disinfectant and deodorant There is an official *Liquor Acidi Chromici* used sometimes as a caustic, the lotion employed as a local application to ulcers, etc., has a strength of 1 in 40 The other official form of chromium is Potassium bichromate (*Potassi Bichromas*,  $\text{K}_2\text{Cr}_2\text{O}_7$ ), it has an antiseptic and caustic action, and has been used in cases of gastric ulcer, it is best given (in doses of  $\frac{1}{16}$ th to  $\frac{1}{32}$ th of a grain) in pill form compounded with kaolin, or in capsules, for it makes explosive compounds See also TOXICOLOGY (*Irritants, Chromium*), PHARMACOLOGY, PRESCRIBING

**Chromocytometer**.—A colour test for estimating the amount of hæmoglobin present in the blood (Buzzozero) See HÆMOGLOBINOMETER (*Haldane's*)

**Chromogens**.—Substances which do not colour the fluid (e.g. the urine) in which they are till it has stood some time, or till an oxidising agent is added See PIGMENTS OF THE BODY AND EXCRETA (*Urobilin*)

**Chromophanes**.—The "colouring matters of the inner segments of the cones of the retina of animals where they are held in solution by a fat" (*Syd Soc Lex.*) See PIGMENTS OF THE BODY AND EXCRETA (*Lipochromes*)

**Chromophile Substance**.—A material, generally in the form of granules, staining markedly with aniline dyes, and existing in nerve cells and their dendrites, but not in the axon, Nissl's granules

**Chromoproteid**.—A simple proteid in combination with a pigment (e.g. hæmoglobin, hæmocyamin, etc.)

**Chromopsia**. See CHROMATOPSIA; but the name has been specially given to cases in

which on account of hyperæsthesia of the retina there are subjective sensations of light in the form of white or coloured clouds (*Hack Tuke*)

**Chromosomes.**—The larger threads of chromatin material which form, during mitotic cell division, when the nuclear membrane disappears

**Chronic.**—Long-continued, *e.g.* a lingering illness, opposed to ACUTE (*q v*)

**Chronotropic Fibres.**—The nerve fibres which affect the rate of contraction of the heart (*Engelmann*) See BATHMETROPIC

**Chrysarobinum.**—Chrysarobin is a brownish yellow powder obtained from *Ilex Powder (Araroba)* by extracting with hot chloroform. It contains chrysarobin ( $C_{10}H_{26}O_7$ ) and chrysophanic acid ( $C_{17}H_{14}O_5(OH)_2$ ). In the form of the *Unquentum Chrysarobini* it is used as a parasiticide in ringworm, etc., but it stains the linen, and it is better, therefore, to apply it as a non-official *pigmentum* (chrysarobin, 1 part, solution of gutta-percha, 9 parts), which does not stain. It is too irritating to the gastrointestinal tract to give internally. See DRUG Eruptions (*Pimentary*), PHARMACOLOGY, PRESCRIBING, PSORIASIS (*Local Treatment*).

**Chrysophan.**—A bitter glucoside obtained from rhubarb and senega ( $C_{16}H_{18}O_8$ ), under the action of acids it breaks up into sugar and chrysophanic acid ( $C_{17}H_{14}O_5$ ), a substance known also as *their acid*, *rhubarbaric acid*, *rhubarbarin*, and *rumicin*. By reducing chrysophanic acid, chrysarobin may be obtained

**Chthononology.**—The science dealing with the geographical distribution of diseases (*χθών*, the soil, *vóros*, disease)

**Chthonophagia.**—Dirt-eating

**Churrus.** See CHARAS, CANNARIS INDICA

**Chvostek's Symptom.**—A state of hyperexcitability of muscles and motor nerves, so that mechanical stimulation, such as a tap, will produce muscular contraction. See TETANY (*Motor Symptom*)

**Chylangioma.**—A lymphangioma, occurring sometimes in the mucous membrane of the small intestine and stomach

**Chyle.**—The milky-looking fluid which is carried off from the small intestine (during digestion) by the lacteals or lymphatics, a variety of lymph containing 6 per cent of fat. See ASCITES (*Character of Fluid*), LYMPHATIC SYSTEM, PHYSIOLOGY, PHYSIOLOGY, THE BLOOD (*Lymph*)

**Chylecchysis.**—An excessive secretion of chyle.

**Chylocele.**—An accumulation of chyle in the tunica vaginalis. See FILARIASIS (*Pathogenic effects, Chylocele*)

**Chyloderma.**—Enlargement of the scrotum and inguinal glands with dilatation of the lymphatics of the skin, which form vesicles containing milky fluid in which filariæ can often be found, lymph-scrotum. See FILARIASIS (*Lymph-Scrotum*)

**Chylopoiesis.**—The formation of chyle. See DIGESTION (*Intestinal Secretion*), PHYSIOLOGY, BLOOD (*Lymph*)

**Chylothorax.**—An accumulation of chyle in the thoracic cavity, *e.g.* from rupture of the thoracic duct

**Chylous Ascites.**—The presence of chylous fluid in the peritoneal cavity from rupture or blocking of the thoracic duct or lacteals. See ASCITES (*Causation*)

**Chyluria.**—A milky or chylous condition of the urine, usually due to the presence of filariæ in the blood. See FILARIASIS (*Pathogenic effects, Chyluria*), URINE, PATHOLOGICAL CHANGES (*Lipuria and Chyluria*)

**Chyme.**—The food after it has been subjected to gastric digestion and before it has been acted on by the bile, the pancreatic, or the intestinal secretions. See DIGESTION AND METABOLISM (*Gastric*), PHYSIOLOGY, FOOD AND DIGESTION (*Stomach*)

**Chymosin.**—A ferment or enzyme which coagulates albumin, *e.g.* the casein of milk, rennin, pepsin

**Cibisitome.**—An instrument for lacerating the capsule of the lens in the early part of the cataract operation (*Petit-Rauel*), the name is derived from *σίστις*, a pouch, and *τομή*, section (*Syn. See Lex*)

## Cicatrices.

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See BURNS AND SCALDS (*Treatment, Results of Burns, etc.*), EYELIDS, AFFECTIONS OF (*Entropion and Ectropion*), INTESTINE, DISEASES (*Ulcers, Cicatrizations*), INTESTINES, SURGICAL AFFECTIONS OF (*Strictures from Ulcers*), LIVER, DISEASES OF, SYPHILIS (*Tertiary, Gummata, and Cicatrices*), MENSTRUATION AND ITS DISORDERS (*Retention of Menses, Vaginal Atresia*), UTERUS, MALFORMATIONS OF (*Stenosis*)

CICATRICES or SCARS are new formations of connective tissue which develop in place of losses of substance

Ordinarily, however, the terms are applied to superficial formations of "scar tissue" which are covered with newly developed epidermis.

A healthy cicatrix has a smooth, more or less glossy surface. In its earlier stages it is of a pinkish colour, due to the persisting vascularity of its connective tissue, later it becomes white, dense, and much less vascular. It differs from true skin in being less elastic and in possessing no surface furrows, hair follicles, sebaceous or sweat glands. The surface shows slight longitudinal ridges, caused by contraction.

The size of a scar depends on the amount of normal tissue which has to be replaced.

A When healing takes place by *first intention*, the resulting cicatrix is "*linear*," and in time it tends to become less and less apparent.

Under the microscope a transverse section shows a narrow, non-vascular band of connective tissue covered by epithelium, and with its elements arranged parallel to the surface.

B When healing takes place by *second intention*, the resulting cicatrix has a more widespread surface. Clinically, its characters depend on the nature of the preceding granulation tissue.

Microscopically, the cicatrix of a wound which has healed by second intention shows on the surface a thin covering of epidermis. This covering is indented from beneath by papillary tufts, which are simply the organised vascular tufts of the preceding granulation tissue. They contain no tactile end-organs, the connective tissue forming them is denser and less vascular than in the case of the papillæ of normal skin, and they tend in time to become more flattened. Below this is fibrous connective tissue, arranged in interlacing bundles, more or less vascular, and more or less cellular, according to the age of the scar. Unna states that elastic tissue may in time be re-formed in the scar. At the margin of the cicatrix its structure rather rapidly merges into that of the surrounding normal tissues.

Cicatrices are divided into three varieties—

1 Normal or flat cicatrices, whose level is the same as that of the surrounding skin.

2 Atrophic cicatrices, in which over-contraction of the fibrous tissue has taken place, and the surface is retracted below the level of the surrounding skin.

3 Hypertrophic cicatrices, where in consequence of over-reaction, or long continuance of the healing process, an excess of scar tissue has been formed, so that the level of the cicatrix is higher than that of the surrounding skin.

Treatment of such cicatrices is only necessary when they cause disfigurement, particularly on exposed surfaces. Measures directed towards keeping up their vascularity, such as *massage* or *painting with iodine*, tend to induce a degree of resorption, or if the surrounding skin is lax, the scar may be excised and the edges accurately apposed with the view of substituting a linear cicatrix.

#### PATHOLOGICAL CONDITIONS OF CICATRICES

1 **WEAK CICATRICES** are thin, shining, reddish, easily wrinkled and cracked, prone to break down and ulcerate in the centre, the friction of the clothes being sufficient to cause this.

They result when the original wound has been very extensive and contraction has been incomplete during the healing process. Hence they occur after severe burns or scalds, particularly if these have affected the tissues deeply, but a smaller cicatrix when it is adherent to bone may also show a weak tendency.

Certain constitutional conditions, as tuberculosis (scrofula), anaemia, syphilis, scorbutus, conduce to the formation of weak cicatrices. The weakness may be due to defective granulation, or defective epithelial formation (keratinisation).

*Treatment*—Treat locally, as for ulcers. In addition to topical applications, the part must be carefully protected from the slightest injury and the general health attended to, treating the constitutional condition if it can be made out.

If the wound has been extensive, there is less chance of good cicatrization, and a plastic operation is necessary. Thiersch-grafting may not give a good result in such an ulcer, and it is better, after scraping or cutting away all the unhealthy tissue, to transplant a piece of skin from the neighbourhood (*vide* "Skin-grafting").

2 **EXUBERANT, HYPERTROPHIC, DEFORMED CICATRICES**—Cicatrices of ragged wounds, such as are caused by glass, splinters of wood, or nails, are generally prominent and ugly. The scars of tuberculous ulcers are extensive and irregular. Wounds which take a long time to heal by granulation may develop a large hypertrophic cicatrix. Treatment is required only when the scar is on an exposed surface, and then it is for the removal of the disfigurement caused.

When the surrounding skin is tense, operative interference is inadmissible, though some improvement may be brought about by measures, such as massage, painting with iodine, etc., directed to the increase of vascularity in the connective tissue, but if the skin be lax, the scar may be completely dissected out, the edges accurately apposed with buried catgut sutures, and a linear cicatrix substituted for the hypertrophic one.

3 **CONTRACTING CICATRICES, CAUSING DEFORMITY**—Such cicatrices may in themselves be quite healthy, but they cause conditions which become pathological.

(a) *Simple contracting Cicatrix*—The amount of contraction depends on the depth of the original wound. When the whole cutis has been destroyed, the contraction is greater than

when only the papillary layers or part of them are destroyed.

The cicatrix is broad, it is thin and parchment-like, of a light colour, mottled on the surface. The surface is uneven, and marked with ridges. Such a cicatrix may be seen after extensive burns of the front of the neck and thorax, and causes dragging down of the jaw, eversion of the lower lip, and constant dribbling of saliva, which is apt to cause superficial ulceration of the scar. If on the face, they cause ectropion or ankylosis of the jaw, and when in the neighbourhood of the natural orifices have caused their distortion or partial closure.

When occurring in flexures they tend to cause adhesions in the angle, and the parts become firmly connected by a strong prominent cicatrix, the so-called *vicious bridle*. Examples of such cicatrices are seen in the axilla—connecting the arm to the trunk, in the bend of the elbow, causing persistent flexure, or in the hands and feet, causing distortion or adhesion of the digits.

If possible, treatment should be begun early in cicatrization, before contraction has taken place, by splints and extension apparatus.

If cicatrization is complete, treatment usually consists in dividing the cicatrix completely, either transversely or in a V-shaped manner, stretching it and filling up the gap thus caused by skin grafts. In making the incision it is well to remember that important nerves and vessels may be involved in or adherent to the cicatrix. Simple extension, compression, and massage of the cicatrix or painting it with iodine have also been recommended with the view of increasing vascularity and promoting absorption, but such measures are never wholly successful. Hebra recommends subcutaneous injection of a 15 per cent alcoholic solution of thiosinamin, but experience of this remedy is limited.

(b) In the *depressed, adherent scar*, treatment consists in dividing the band of attachment, by means of a tenetome passed in from the edge, and raising the scar. Less active measures, with a view to increasing the vascularity and promoting absorption, are never wholly successful.

When it results from a scrofulous (tuberculous) gland, Adam's operation (*Brit Med Journal*, 1876) may be employed. After freeing the cicatrix from the side, he passes two hare-lip pins, at right angles to each other, under it so as to raise it. After three days, during which the scar tissue has become swollen and succulent, and the space beneath filled with blood clot, he removes them. The scar subsides to the level of the skin without re-forming adhesions.

Cicatrices may also cause deformity by yielding to pressure from beneath, as in those following laparotomies, when ventral hernia may result.

**4 PAINFUL CICATRICES**—Pain may be present in any cicatrix, but is most common in those of amputation wounds. The pain is caused by inclusion of the nerves in, or their adhesion to, the cicatricial tissue. Some authors say that the pain is caused by neuritis dependent on the contraction of the scar.

*Neuralgia of the Cicatrix*.—In this case the pain, which is paroxysmal, is generally due to some constitutional condition affecting a cicatrix in itself healthy. It may persist in spite of operative interference.

*Treatment*.—Free adhesions in case of depressed or adherent cicatrices. If the cicatrix be small, excise it altogether and slide a flap of skin over it. If the cicatrix be extensive, neurotomy, neurotomy, or nerve-stretching may be useful.

Where there is no abrasion of the surface, in the case of a small, painful cicatrix, ointment of aconitine (1 gr to 3i) is of use as a palliative, or, if broad, it may be protected by simple plaster, or emplastrum opii, or may be simply covered with cotton-wool.

**5 DISCOLOURED CICATRICES**.—The cicatrices from old chronic and varicose ulcers have always a permanent brown colour. Large scrofulous cicatrices have an unsightly livid tint.

Foreign matter may be present, such as unconsumed grains of gunpowder in cases where the injury has been caused by an explosion, or in tattooing.

Occasional corneal opacities are due to the presence of reduced silver, where the preceding ulcer has been treated with silver nitrate.

These discolorations occasionally require treatment when on exposed surfaces.

#### KELOID, OR CHELOID

is a comparatively rare fibrous new growth of the skin which usually develops in an old cicatrix. It was first described by Alibert in 1810. Keloid was formerly divided into true or spontaneous and false or cicatricial keloid, but this distinction is no longer generally held, as most of those termed spontaneous probably develop in minute scars, such as are left by acne pustules. Histologically they are identical.

The etiology is practically unknown. Its growth seems to be dependent on some constitutional state, for example, it is very frequently associated with the tuberculous diathesis. It is more common in negroes than in white men, and it may occur in more than one member of the same family. All grades of society are liable to it, both sexes, all parts of the body, and it may occur at almost all periods of life. Morrow states that in his experience it is more common between 30 and 50 years.

Keloid often develops in scars which have been unduly irritated, and it may develop in a healthy scar long after this is fully formed.

In individuals liable to it, it develops after such slight operations as piercing the ear for ear-rings, extirpation of warts, cauterisation, or after leech and insect bites, or on the sites of acne pustules. It may be multiple, as in a case of Schwimmer's quoted by Morrow, in which 105 tumours were counted.

Keloid is a scar-like growth, usually circumscribed and isolated. It may occur as a rounded, firm, pinkish elevation, with irregular or smooth surface, and on section creaks under the knife. Often from the central mass claw-like processes extend in the surrounding skin. As a rule it has the shape of the scar from which it has developed, but occasionally a part only of the scar undergoes the morbid change.

Neumann, Warren, and Kaposi have described the microscopical appearance.

Connective tissue begins to develop from the walls of the small blood-vessels of the cutis. In this way dense fibrous tissue is formed under the epithelium, the bundles being placed chiefly parallel to the surface, though a few small bundles are perpendicular to it. This is more vascular than true scar tissue. The epithelium is thinned over it, and the germinal layer of the rete mucosum becomes irregular. The growing part of the tumour is more cellular than the central older part, which may become very firm, dense, and hollowed on the surface. Frequently dilated veins course over it.

The tendency to recurrence is explained by the development of spindle cells in the adventitia of the vessels—a condition often extending to some distance from the edge of the tumour.

Keloid is usually confined to the dermis, leaving the papillary layers intact. If of a large size it may cause deformity by contraction. It may project any distance from a line to  $\frac{1}{4}$  or  $\frac{1}{2}$  inch above the level of the surrounding skin.

Clinically, keloid starts near the edge of the scar, it may cause no symptoms other than a sense of weight. There are usually, however, subjective symptoms such as itching or sensitiveness on pressure, pain is not common, ulceration and hæmorrhage may take place.

It may develop in a few weeks, or at most a few months, after the exciting cause. Its natural tendency, as of all young fibrous tissue, is to contract after a time and atrophy.

According to Neumann keloid never develops malignancy.

**Diagnosis.**—From hypertrophic cicatrix by its outline, elevation, consistence, colour, and presence of itching or pain, and by the tendency of the latter to become stationary.

The diagnosis from scleroderma and Addison's keloid (morphea) will be discussed under "Scleroderma," and from rhinoscleroma, under "Skin, Benign Tumours."

**Treatment.**—Local treatment is directed to wards decreasing the vascularity of the tumour. Applications like collodion (not flexible), either alone or containing lead acetate, a drachm to five drachms of collodion, are useful. Compresses, strapping, elastic pressure, must be applied with caution, as they are apt to cause sloughing, and are only applicable when the keloid is circumscribed, painless, not greatly projecting, and resting on a bony surface. They are especially contra-indicated in diabetics and delicate patients. Multiple scarifications are of doubtful benefit, either alone or followed by mercurial ointments. In a few cases strong salicylic acid plasters 10 to 20 or 25 per cent have been successful.

Itching is best relieved by regulation of the diet, by an occasional saline purgative, Fowler's liquor arsenicalis, liquor potassæ in large doses, or by frequent tepid bathing, either simple or with addition of carbonate of soda or potash, common salt or bran.

Pain is relieved by ointments of camphor and chloral.

Surgical interference by excision is only justifiable when the surgeon is reasonably certain that he can improve the appearance of the part by substituting a linear scar for the keloid, or at least of lessening its size.

Tendency to recurrence is very marked, and the recurrence is often more exuberant than the original growth. In most cases it is best to leave well alone, and employ only palliative and constitutional remedies.

Excision may be contemplated when the keloid is very unsightly and where there is plenty of free skin surrounding, so that the edges of the wound can be readily apposed without tension. The growth must be completely removed, and buried sutures used, approximation being assisted by strips of gauze fixed to the skin with collodion. Everything must be done under strict asepsis.

**Constitutional Treatment.**—Cod-liver oil, iodide of iron, nourishing diet, are of use where tuberculosis is suspected, mercury and the iodides in syphilis. Good results have been obtained in the treatment by sea bathing.

**MALIGNANT DISEASES OF CICATRICES.**—Sarcomata develop rarely in cicatrices. More commonly, but still rarely, epitheliomata occur. They may develop at any period after the cicatrix has formed. One case is on record where epithelioma developed in an old lady in the scar of a burn which she received when a child of four years.

Epithelioma develops very slowly in the cicatrix, and may not involve neighbouring glands until it grows beyond the cicatrix into normal tissue, in which it develops more rapidly.

Treatment consists in early and complete excision.

**Ciechocinck.** See BALNEOLOGY (Russia).

**Cigarettes.**—Drugs, such as stramonium, may be inhaled in the form of cigarettes, as in asthma See PRESCRIBING, ASTHMA (Treatment), etc

**Cilia.**—The hair-like processes from the free surfaces of cells (e.g. epithelial cells), which move to and fro with a vibratory motion See PARASITES, PROTOZOA, (Ciliated), PHYSIOLOGY, TISSUES (Epithelium, Ciliated), also, the eyelashes See EYELIDS, AFFECTIONS OF

**Ciliary Body.** See IRIS AND CILIARY BODY (Anatomy, Physiology, Diseases), PHYSIOLOGY, SENSES (Vision, Ciliary Processes, Ciliary Muscle) See also ACCOMMODATION (Spasm of), DIPHTHERIA (Complications, Paralysis of Ciliary Muscles); RETINA AND OPTIC NERVE (Congenital Irregularities in Vessels, Cilio-retinal Artery)

**Ciliosis.**—A spasmodic trembling of the eyelids, or the sensation of such, the feeling of "live blood"

**Cimex.**—A bug, the bed bug (*Cimex lectularius*) may by its bite produce a great deal of cutaneous reaction (like urticaria), but some individuals seem to be immune to its poison See STINGING INSECTS

**Cimicifuga Rhizoma.**—The rhizome of the *Cimicifuga racemosa* (perhaps better known as *Actaea racemosa*) is used as a bitter, and also, to some extent, as a heart tonic (raising the arterial tension), there are two official preparations, the *Extractum Cimicifugae Liquidum* (dose, 5 to 30 m), and the *Tinctura Cimicifugae* (dose,  $\frac{1}{2}$  to 1 fl dr), it is employed in the treatment of chorea, rheumatism, dyspepsia, and dysmenorrhoea, the name (*cimicifuga*) literally means "putting bugs to flight," and the *cimicifuga racemosa* is popularly termed bugbane

**Cimiez.** See THERAPEUTICS, HEALTH RESORTS (Rivera, Nice)

**Cinchona.** See QUININE, ALKALOIDS, PHARMACOLOGY, PRESCRIBING, MALARIA, etc

**Cinchonine.**—An alkaloid ( $C_{19}H_{22}N_2O$ ) obtained from cinchona, isomeric with *cinchonidine* and with *cinchonine* See QUININE, etc

**Cinchonism.**—Poisonous symptoms (tinnitus aurium, deafness, impairment of vision, headache, dilatation of pupils, etc) due to use of quinine in large doses or to an idiosyncrasy of the individual

**"Cinder-sifting Movement."**—In cases of movable or floating kidney, a tilting movement of the organ may take place in the plane of the loins by which the convex border of the kidney turns forwards, this is termed

the "cinder-sifting movement" See KIDNEY, SURGICAL AFFECTIONS (Movable and Floating Kidney)

**Cinematograph.** "A contrivance by which a series of instantaneous photographs taken in rapid succession can be projected on a screen with similar rapidity, so as to give a life-like reproduction of the original moving scene" (Murray, *N.E.D.*), the cinematographic (or kinematographic) method has been used for the representation of surgical operations and of medical symptoms (ataxia gait, etc), and may yet be employed for embryological processes, etc

**Cinesitherapy.** The treatment of diseases by means of natural or artificial movements of the body, e.g. by Klapp's four-footed progression movements in spinal curvature, etc

**Cinnabar.**—Red sulphide of mercury occurring as an ore in Spain, California, Peru, and elsewhere, from it mercury is obtained by roasting and distilling

**Cinnamic Acid.**—An acid ( $C_9H_8O_2$ ) obtained from the balsams of tolu and Peru, or from storax, it can be artificially made by heating 2 parts of benzaldehyde with 3 of acetic anhydride and 1 of sodium acetate (Perkin's reaction) From it *cinnamene* ( $C_8H_8$ ), a volatile oil, can be got, while from *cinnamic aldehyde* ( $C_9H_8O$ ) can be obtained *cinnamyl-acrylic acid* ( $C_{11}H_{10}O_2$ ) and *cinnamylangelic acid* ( $C_{13}H_{14}O_2$ ) *Cinnamene* is an oil ( $C_9H_8O_2$ ,  $C_7H_7$ ) contained in the balsams of tolu and Peru, and obtained artificially by heating sodium cinnamate with benzyl chloride, it is benzyl cinnamate See IMMUNITY (Treatment of Tuberculosis by cinnamic acid), LUNG, TUBERCULOSIS OR (Treatment, Specific)

**Cinnamon.**—The dried inner bark of *Cinnamomum zeylanicum* is official, and is known as *Cinnamomi Cortex*, it has a fragrant odour, and the well-known taste It contains the official oil (*Oleum Cinnamomi*), tannin, sugar, and gum, it is contained in various powders and tinctures, and its dose is from 10 to 20 grains The official preparations of the cortex are the *Aqua Cinnamomi* (dose, 1 to 2 fl oz), the *Pulvis Cinnamomi Compositus* (dose, 10 to 40 grains), and the *Tinctura Cinnamomi* (dose,  $\frac{1}{2}$  to 1 fl dr), the *Aqua* and the *Pulvis* are contained in various other pharmaceutical preparations The *Oleum Cinnamomi* consists chiefly of cinnamic aldehyde ( $C_9H_8O$ ), a terpene, and eugenol ( $C_{10}H_{12}O_2$ ), it is given in doses of  $\frac{1}{2}$  to 3 m, and its official preparation, the *Spiritus Cinnamomi*, has a dose of 5 to 20 m Cinnamon is used for flavouring medicines, the bark has also astringent properties, and the oil is a stomachic and carminative. See also PHARMACOLOGY, PRESCRIBING, etc

**Cion**.—In compound words *cion* (Gr. *κίον*) means the uvula, as in *cionoptosis*, relaxation of the uvula, *cionotomy*, cutting the uvula, *cionotome*, etc.

**Circinate**.—Rounded.

**Circle of Haller**.—A vascular plexus on the sclerotic, near the entrance of the optic nerve, formed by branches of the short ciliary arteries, and named on this account *circulus arteriosus Halleri*, the *circulus venosus Halleri*, or *circulus venosus mammae*, is that formed by the veins round the nipple

**Circle of Mascagne**.—The name given to the circular arrangement of capillaries in the region of the zonule of Zinn in the fetus

**Circle of Willis**.—The circular arrangement of arteries at the base of the brain (*circulus arteriosus Willisii*), into the composition of which enter the anterior and posterior cerebral and the anterior and posterior communicating arteries See BRAIN, PHYSIOLOGY (Blood Supply)

**Circular Insanity**.—Insanity in which a dull and depressed stage alternates with one of excitement

**Circulation**. See HEART, MYOCARDIUM, AND ENDOCARDIUM, ARTERIES, DISEASES OF, CAPILLARIES, DISEASES OF, VEINS, DISEASES OF See also ANEURYSM, CHILDREN, CLINICAL EXAMINATION, PHYSIOLOGY, CIRCULATION, FETUS AND OVUM, DEVELOPMENT (*Fetal Circulation, Placental Circulation*), LYMPHATIC SYSTEM, PREGNANCY, PHYSIOLOGY (*Changes in Circulation*), PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Cardio-Vascular*), PULSE

## Circumcision.

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See also VENEREAL DISEASE (*Soft Sores, Complications, Phymosis*), MICTURITION (*Incontinence, Retention*), SPERMATORRHEA, URTICARIA, DISEASES (*Gonorrhoea, Complications, Phymosis*)

CIRCUMCISION is the operation for the removal of the prepuce in its whole circumference. Its origin is obscure. As a religious ordinance it dates from the time of Abraham, but there are proofs of an earlier Egyptian origin, while it was undoubtedly practised in early times in Central America, New Zealand, and the South Sea Islands. At the present day it is not only a Jewish ritual performed in infancy, but is customary at puberty among Mohammedans, and prevails in some parts of Central Africa.

Apart from the question of religion and custom, one may say, generally, that circumcision is indicated where there is a condition of phymosis,

that is, such a narrowing of the prepuceal orifice where the cutaneous and mucous membranes meet, that retraction and reposition are difficult or impossible. Difficulty in retracting the prepuce from epithelial adhesions to the glans is normal at birth, and is not phymosis, and only becomes so when natural or artificial means fail to relieve the condition. Operation is, therefore, only indicated when the normal process of retraction is delayed, and the orifice narrowed by slight recurring attacks of balanoposthitis, and more especially if complications arise which experience has proved to depend upon this condition. The phymosis may be partial or complete hypertrophic when the outer layer of the prepuce is elongated, atrophic when tightly stretched over the glans, inflammatory, oedematous, or senile.

The complications arising from phymosis are many; they may take the form of urinary troubles, local inflammation and irritation, a frequent combination of the two, and maldevelopment of the penis and glans. Of urinary troubles there may be frequency of micturition, straining and pain, enuresis, retention, incontinence, and overflow, dilatation of the bladder, ureters, and kidneys, with, as a consequence, pro-lapsus ani, hernia, hæmaturia, uræmia, cystitis, and stone. From local inflammation and irritation arise pain, swelling, oedema, mucopurulent discharge with increase of the phymosis, excoriation, masturbation, seminal emissions, and other disturbances of the sexual functions, leading to nervous troubles, gastralgia, palpitation, hypochondriasis, and it is even said simulated or real disease of the hip-joint, epilepsy, and brain affections. A combination of the two naturally aggravates both, while later in life maldevelopment of the penis and glans, difficulties in coitus, and the liability to venereal disease and epithelioma have to be considered. Although all conditions of true phymosis are really acquired, as proper treatment in infancy would have prevented them, still for convenience, those occurring in children whose foreskins have never been retracted are termed congenital, while those which appear later in life, when the prepuce has previously been easily retracted, are called acquired. Acquired phymosis can only occur when the prepuce is long, as it normally is in 33 per cent of adults. The causes are usually inflammatory, from gonorrhoea, chancres, eczema, or herpes, but senile phymosis depends on atrophy of the glans and limb of the penis, with chronic irritation.

Should one or several of these conditions exist, the next question to consider is when is operative interference necessary, and regarding this the most varied views prevail.

According to some, every long prepuce, whether it can be retracted or not, demands operation, as it may at a subsequent date lead to trouble. The other view is, that, as in the majority of cases,



even when in childhood, there is a slight stenosis of the orifice, this condition disappears naturally with growth and suitable treatment, it is always wise to wait until complications arise, and to make sure that these depend on the condition of the prepuce before interfering.

Of these two views the latter certainly appears to be the more sensible.

It is a safe rule, therefore, that all conditions of phimosis, in which retraction and reposition are difficult or impossible, and which give rise to symptoms, should be subjected to operation. The operation, whatever its nature, should be thorough, and lead to a sufficient and permanent widening of the preputial orifice, with complete separation of adhesions, partial operations, such as insufficient dorsal incision, frequently causing increased phimosis through cicatrisation. The form of operation to be preferred varies, depending upon which variety of phimosis is present.

*A. In the atrophic form*, when the prepuce is stretched over the glans, and the outer layer does not project, a dorsal incision is usually sufficient.

The patient having been prepared, a general anæsthetic is administered, though local anæsthesia, such as that produced by the local injection of cocaine, may in exceptional cases be employed. In cases where masturbation has been practised, it is recommended by some that no anæsthetic should be used, the moral effect of the pain being supposed to have a deterrent influence.

In youths and adults a red rubber catheter should be tied round the root of the penis to prevent hæmorrhage, in infants this is inconvenient and unnecessary.

Elaborate disinfection of the surrounding parts may be dispensed with, the penis being drawn through a small hole in the centre of a piece of lint, sterilised or moistened with warm 1 in 10 carbolic solution, and the surrounding parts covered with sterilised towels.

The penis being fixed, and the foreskin firmly retracted with the left hand, a grooved director is carefully introduced between the prepuce and glans on the dorsal aspect, and along this the blunt blade of a small pair of scissors is passed as far back as possible, and the prepuce divided exactly in the middle line. The edges at once gape, so as to form an almost transverse incision, the inner layer being divided half-way up, the outer layer somewhat higher. By further traction on the skin of the penis, the outer layer is drawn back as far as possible, and from the angle of the inner layer a small triangular flap is cut with its base at the corona, and its edges meeting at an angle of  $60^\circ$ , its length being about half an inch in the adult and half that in infants. Irregularities of the edges of the inner layer are removed with scissors, and the angular extremities of the first cut rounded off. Any bleeding points having been twisted or ligatured,

all adhesions between the prepuce and glans are separated, slight ones by means of a blunt probe, those that are firmer are cleared all round and then divided close to the glans, so as to wound the inner layer of the prepuce as little as possible, and thus prevent œdema, if necessary, any larger wound of the inner layer is closed with a fine catgut suture.

The interior of the prepuce is then cleaned with a 1 to 40 carbolic lotion to remove smegma, and the wounds sutured. This is best done with a continuous suture of fine catgut so as not to require subsequent removal. One commences by adjusting the apex of the little triangular flap of the inner layer into the angle of the wound in the outer layer. This method is recommended by Rosen as forming a skin edge at the apex of the incision, and thus preventing the healing of the original wound, with the formation of a firm, immobile cicatrix, or even a recurrence of the phimosis.

Local after-treatment consists in keeping the part as much at rest and as aseptic as possible. A good method consists of dusting on a little powdered crystallised iodoform, and surrounding the part with a narrow strip of several layers of iodoform gauze, while over this a narrow carbolic gauze bandage is applied with moderate firmness up to the root of the penis, this acting as a kind of splint. Care must be taken to prevent the dressing as far as possible from becoming soaked with urine. It may be left on for several days unless the part be painful, being occasionally moistened with weak carbolic oil if it becomes wet. Another method recommended is to keep the part covered with a small strip of lint, kept moistened for the first twenty-four hours with a mild antiseptic lotion, this is then renewed and allowed to dry on.

Complete primary union is rare in children, but not uncommon in adults. Any point that remains unhealed is best treated by being smeared with a little soft boracic ointment.

The patient should be kept in bed for two days, and then allowed to lie on a sofa, but he is not to move about for a week. Bromide of potash may be required in adults if erections be troublesome.

*B. In the case of hypertrophic phimosis* which occurs so frequently in children, dorsal incision is unsatisfactory, leading to œdema and an inelegant result. Circumcision is, therefore, to be preferred. This operation, although it has undergone innumerable modifications, differs little in essentials from that originally performed and still practised as a religious rite by the Jews.

A point on the foreskin about a quarter of an inch in front of the corona is grasped obliquely



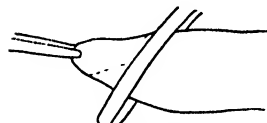
from above downwards, and from behind forwards with long dressing forceps, or other special forceps, the obliquity making the resulting opening elliptical, and thus larger. Care must be taken that the glans is pushed back and is not included in the grip of the forceps. The anterior portion of the prepuce in the grasp of the forceps is then cut off by one sweep of a sharp bistoury.

The outer layer retracts to a considerable extent, and care is, therefore, necessary not to include too much in the forceps, otherwise the limb of the penis is apt to be left devoid of skin, this leading to delay in healing and subsequent discomforts during erections.

The inner layer still remains more or less enclosing the glans, it is to be divided by means of scissors in the mid-dorsal line almost to the level of the corona, or Roser's triangular flap may again be employed with advantage. The angles are then trimmed off up to the frenum, leaving a small fringe of mucous membrane surrounding the corona. Bleeding having been carefully arrested, the edges are brought together by means of a continuous or interrupted fine catgut suture, the former being preferable, but in the case of infants sutures are frequently unnecessary. If the triangular flap has been employed, it is well for exactness of adjustment to make a small mid-dorsal snip in the outer layer to receive it. The after-treatment is as formerly described. Should the wound not heal by first intention, as is usually the case at the frenum, warm baths and a mild antiseptic ointment are grateful and promote healing.

While these are the general principles upon which the operations should be conducted, there are, however, certain points to which further attention must be directed.

*First*, As to the employment of the elastic band to prevent hæmorrhage.



Davies Colley's Modification

move either too much or too little of the prepuce.

*Second*, The forceps having been applied, it is usual to cut behind them. Davies Colley, however, strongly recommends that the incision should be made in front of them, and the distal portion of the prepuce having been made tense, that the lower part of the incision should be made obliquely from behind forwards, so as to form a triangular flap of the middle of the under surface of the prepuce, which, he says, fits more accurately into the triangular gap with its apex

of the elastic band. When this is used, care must be taken before application to mark on the skin the exact position of the corona, otherwise one is apt to remove

at the stump of the frenum, which is formed on removal of the inner layer.

*Third*, When an elastic band has been employed in adults, primary union is frequently obtained and time saved by tying any obvious vessel, suturing and applying the dressing and bandage with moderate pressure before removal of the band. The subsequent swelling prevents further bleeding, but experience is necessary, as a too tight application of the bandage before removal of the elastic band may cause such subsequent pressure as may lead to slight sloughing of the edges of the wound.



*Lastly*, As to the question of operation in the case of gonorrhœa and inflamed hard, soft, and phagedenic chancres, the fear of infection and inflammation of the whole wound has frequently prevented this from being done when otherwise advisable, this, however, rarely occurs if the raw surfaces are well washed with carbolic lotion, any ulcerated surface not capable of removal being first dried and then touched with fuming nitric acid and iodoform rubbed in. In the case of great swelling and inflammation, a dorsal incision followed by disinfection and subsequent removal of the redundant flaps by means of scissors, no sutures being employed, often gives a better result than the more classical operation.

Circumcision is usually devoid of risk, but fatal cases have been recorded from cellulitis, erysipelas, tetanus, and hæmorrhage, while infection from syphilis and tubercle are not unknown as the result of ritual circumcision among the Jews.

**Circumclusion.**—A variety of acupuncture in which the pin is passed under the vessel and a wire loop over it.

**Circumduction.**—A sweeping movement of a limb whereby it is made to describe a cone with its apex at the proximal extremity of the limb.

**Circumferences.** See LABOUR, PHYSIOLOGY (*Fœtal Head Circumferences*).

**Circumflex Nerve.** See NERVES, PERIPHERAL (*Affections of Special Nerves, Circumflex*).

**Circumstantial Evidence.**—Evidence inferred from circumstances affording a presumption but not a certain proof, opposed to *direct* or *positive*. See MEDICINE, FORENSIC (*Wounds and Injuries*).

**Circumvallate.**—Surrounded with a rampart or raised border, e.g. a circumvallate placenta, or one in which the fetal membranes are attached at some distance from the periphery, leaving a circular ridge all round.

**Cirrhosis.**—The morbid change in an organ or structure by which it becomes firmer in consistence and, as a rule, smaller in size, it is produced mainly by hyperplasia of the connective tissue of the organ or part. See LIVER, DISEASES OF (*Biliary Cirrhosis*, *Portal Cirrhosis*), LUNGS, PNEUMONOKONIOSIS (*Pathological Anatomy*), NEPHRITIS (*Renal Cirrhosis*)

**Cirsocoele.**—A varicose state of the spermatic veins forming a swelling, a varicocele, the word is derived from *κίρσος*, a varia, and *κύλη*, a swelling. The condition may be combined with hydrocele (*cirsychochecele*)

**Cirsoid Aneurysm.**—A swelling composed of enlarged and dilated arteries. See ANEURYSM (*Arterio-venous Intercommunications*)

**Cirsomphalos.**—A varicose condition in and around the umbilicus, caput medusæ (q v)

**Cirsophthalmia.**—Ophthalmia associated with a varicose condition of the conjunctival blood-vessels

**Cirsotomy.**—The operation of excision of varices

**Cissampelos.**—The dried wood of *Cissampelos javanica*, official in the Indian and Colonial Addendum to the B.P., it is used in cystitis and allied states, and it is given either as the *Decoctum Cissampeli* (dose,  $\frac{1}{2}$  to 2 fl oz) or as the *Extractum Cissampeli Liquidum* (dose,  $\frac{1}{2}$  to 2 fl dr) See AGROPHYRUM

**Cisterns.**—Cisterns for storing water for drinking purposes should be made of galvanised iron or slate, not of lead (see TOXICOLOGY, *Chronic Lead Poisoning*), they should be ventilated, should have an overflow pipe passing directly into the open air, and should be easily accessible for purposes of cleansing and inspecting, they should hold about three days' supply of water, a water-closet should have a small cistern of its own (which may be made of lead), which should empty completely each time, and be supplied from the large cistern. See WATER

**Citarin.**—An antipyretic and antirheumatic preparation (*Merk*), being sodium anhydromethylene citrate, used in doses of 8 to 20 grains in rheumatism, gout, neuralgia, etc

**Citric Acid.**—Acidum citricum ( $\text{H}_3\text{C}_6\text{H}_5\text{O}_7 \cdot \text{H}_2\text{O}$ ) exists in the juice of many fruits, and is usually got from the lemon or lime, it is contained in the *succus limonis* and *syrupus limonis*, and, as the citrate, in preparations of potash of iron and ammonium, and of iron and quinine, it may be given as a refreshing drink in fever (lemonade), or as an antiscorbatic (lime or lemon juice), the dose of the acid is 5 to 20 grains

**Citrine Ointment.**—Unguentum hydrargyri nitratis. See MERCURY.

**Citrosis.**—Depraved appetite, pica

**Civet.**—An unctuous substance with a musk-like odour, obtained from the pouch lying between the anus and the genital organs of the civet cat (*Fiveria civetta*), and formerly used as an antispasmodic

**Civiale's Method of Internal Urethrotomy.**—Internal urethrotomy in which a concealed knife is passed through the structure, which is divided from behind forwards. See URETHRA, DISEASES OF (*Internal Urethrotomy*).

**Civil Incapacity — Capacity (Mental) for Civil Contract, etc.**

—It may be stated generally that a person while insane is legally incapable of entering into a contract, he is in the legal sense incapable of giving the consent which is essential thereto. Consequently he cannot enter into the contract of marriage, nor can he execute a will. Moreover, the management of his property may be removed entirely from his control.

*What constitutes Legal Insanity*—It is not possible to formulate any criterion which would be generally applicable to the varied cases which arise. To generalise in the matter would be to mislead. The question of insanity in such cases is always the question of capacity intelligently to do the particular act. The late Lord President Inglis (in the Scottish case of Morrison, 1862, 24 D at p 631) said to the jury: "I am not going to give you any definition of insanity, and I am not even going to define to you what legal capacity is in a question of this kind, because I may tell you at once that the question whether a man is in such a state of mind as to be capable of executing a deed of this kind is a question of fact, and not of law."

The test of his capacity to execute such a settlement may be very reasonably stated with reference to the nature of the settlement itself, but it cannot possibly be stated without reference to the settlement, because a man may have strength of mind—power of intellect sufficient to enable him to do one thing, to make one kind of mental exertion—and yet he may be totally incapable of making another. A man may be so far weakened in his mind as not to be able to follow a difficult process of reasoning . . . and yet be perfectly capable of making a simple destination of his property. The two things are totally different. You can easily understand that a man may not be capable of very long-sustained mental exertion, and yet may be quite capable, reasonably and fairly, of saying, I want my estate to go to A B, or I want to disinherit my heir and to leave my estate to an hospital. That is not a very complex idea, and if a

person is capable of distinctly understanding what it is that he is doing—is capable of expressing that purpose in intelligible language, and is capable of understanding the consequences and effects of what he does—then he is capable of making such a settlement, and it is vain to go about scientific definitions or anything of the kind" (*see also* Erskine, J., in *Harwood*, 1840, 3 Moo P. C. 282).

A deed disclosing no trace of incapacity or insanity may nevertheless spring from an insane belief or delusion, but in establishing this to the effect of invalidating the deed, it is necessary to prove not merely the groundlessness of the belief, but also the impossibility of its being entertained by any sane mind. The existence of a disordered belief upon some extraneous point, having no connection with the subject matter of a settlement, is not necessarily fatal to the deed. In a recent case the House of Lords (reversing a judgment of the Scottish Courts) held that these alleged facts, if established, would be sufficient to invalidate, on the ground of mental incapacity, the will of a testator who directed the greater part of his large estate to be employed in promoting total abstinence and preventing the spread of Roman Catholicism. It was stated that upon these topics he was subject to insane delusions, and believed that he had an imperative duty to devote his pecuniary resources to these objects in consequence of commands which he had received from the Deity by direct communication upon various occasions, and these insane delusions dominated his mind and mastered his judgment to such an extent as to render him incapable of making a reasonable and proper settlement of his estate (*Hoppe*, 1898, 1 Fraser, H. L. 1, *see also* Lord Penzance in *Smith*, 1867, L. R. 1 P. & D. 398).

It is necessary, then, that a testator should have sufficient memory to recall the amount and character of the property of which he is possessed, and to remember the several persons who may be said to have claims on him. Along with this memory, he must also have his judgment reasonably clear and free from morbid and insane ideas, so as to be able to decide as to these claims.

The question of the mental capacity of a person to enter into a particular contract or to execute a particular will is frequently combined with allegations of undue influence exercised by those who benefit by this act. The inquiry is, then, of course, pointedly directed to determining whether he was facile, unduly liable to persuasion, easily misled to wrong views and feelings,—how far incapable of resisting pressure.

This incapacity to enter into civil contracts, etc., exists only when and so long as the person is insane. If he recovers his sanity permanently, or even temporarily, his contracts and deeds

are valid. A will executed in a lucid interval, even by a person then confined in an asylum, is perfectly valid (*see* Nisbet's *Treatise* 1871, p. 9; Macpherson's *Reports*, p. 937). In such a case the rationality of the document itself is regarded as an important piece of evidence in favour of sanity. The burden of proof is shifted—that is all. Whereas a man is presumed sane until he has been proved to be insane, a person once proved to be insane is presumed to be insane until his recovery is proved.

**Moral Insanity.**—It has been judicially laid down in England (by Sir H. J. Fust in *Frere*, 1846, 1 Rob. E. R. pp. 412, 446) that moral insanity, unaccompanied by delusion, does not invalidate a will. But this can scarcely be said to be recognised in modern practice. There can be little doubt that proof of the insane perversion of the moral feelings would at least be held sufficient to invalidate a will (*see* Wood Renton on *Lunacy*, p. 55).

**Civilization.**—The advance or development of human society from a less to a more complicated condition of affairs, from a lower to a higher grade of complexity, this is believed (from a medical standpoint) to increase the frequency of insanity and cancer, and to make the process of childbirth more difficult. *See* INBORN, EPIGENESIS.

**Civitavecchia.** *See* BALNEOLOGY (*Italy, Sulphur Waters*).

**Cladotrix Dichotoma.**—A species of cladotrix, one of the genus of Schizomycetes, the name is derived from *κλάδος*, a branch, and *ὄψις*, hair, in reference to the pseudo-branched filaments of which the growth is composed. *See* MICRO-ORGANISMS (*Bacteria, Schizomycetes*), TEETH (*General Bacteriology of Mouth*).

**Clairvoyance.**—The supposed state of mind of a person who can, it is said, see mentally, when under the influence of animal magnetism, places and persons and things he has never seen before, and give a correct description of them, this supposed power has been alleged to be found to be of use in medical diagnosis.

**Clamp.**—An instrument for grasping and compressing firmly structures and tissues, e.g. an artery to stop hæmorrhage. *See* ANGIOTRIPE, etc.

**Clap.** *See* URETHRA, DISEASES OF (*Gonorrhœa*).

**Clapotage.**—The splashing sounds heard on auscultation over a dilated stomach.

**Clarke's Column.**—The group of nerve cells lying at the inner angle of the posterior horn of grey matter in the spinal cord, the columna vesicularis.

**Clark's Process.**—The method of moving the hardness of water by adding hydrated calcium oxide, which throws down the carbonate of lime, which gradually subsides and carries down suspended matters with it 1 oz per 100 gallons is added for every degree of temporary hardness, *i.e.* for each grain per gallon

**Claudication.** -- Lameness, as in hip-joint disease as an early symptom, also when due to spasm, as in anemia and progressive arterio-sclerosis, when it has been named *claudication intermittens* (See SPASM)

**Claustromania.**—The insane impulse to take to "human burrows" or enclosed spaces See INSANITY, NATURE AND SYMPTOMS (*Impulse and Obsession*)

**Claustrophobia.**—The fear of being in a closed space, as contrasted with *agoraphobia* (*q.v.*), the fear of open spaces See INSANITY, NATURE AND SYMPTOMS (*Impulse and Obsession*)

**Claviceps Purpurea.** See ERGOT, TOXICOLOGY (*Ergotism*)

**Clavicle.** See BRACHIAL PLEALS, SURGICAL AFFECTIONS OF (*Tears and Contusions in Fracture of Clavicle*), FRACTURES (*Clavicle*), SHOULDER, DISEASES AND INJURIES OF (*Dislocation and Fracture of Clavicle*), SHOULDER, DISEASES AND INJURIES (*Excision of Clavicle*), STERNO-CLAVICULAR JOINT (*Injuries and Diseases*), TERATOLOGY (*Absence of Clavicle*)

**Clavus.**—A corn or hardened projection of the epidermis due to friction or intermittent pressure See CORNS, TUBER DORSALIS (*Perforating Ulcer of Foot*)

**Clavus Hystericus.**—The painful sensation as if a nail were being driven into the head, *clon hystérique* See HYSTERIA (*Hyperæsthesia, Cephalalgia*)

**Claw Hand.**—The deformity of the hand ("main en griffe") due to progressive muscular atrophy, and brought about by contraction of the extensor and flexor muscles, and atrophy of the thumb muscles, the interossei, and the lumbricales See PARALYSIS (*Progressive Muscular Atrophy*)

**Cleft.**—A fissure, groove, or narrow space between two projecting surfaces, *e.g.* cleft palate, facial or genial cleft, cleft of the nates, vulvar cleft, cleft sternum, etc See CHEEK, FISSURE OF, CHEST, DEFORMITIES OF (*Cleft Sternum*), EMBRYOLOGY (*Branchial Clefts*), PALATE (*Cleft Palate*), PALATE (*Congenital Malformations of the Mouth*)

**Cleido.**—In compound words *cleido-* signifies relating to the clavicle, *e.g.* *cleido-brachial*,

*clavicular*, relating to the clavicle and the arm, *cleido-cervical*, relating to the clavicle and the neck, *cleido-hyoid*, relating to the clavicle and the hyoid bone, and *cleido-mastoid*, relating to the clavicle and to the mastoid process of the temporal bone

**Cleidotomy.**—Division of one or both clavicles, an accessory operation in cases of en'chylæmia, in order to diminish the bisacromial diameter of the fetal trunk and so facilitate delivery of the shoulders See LABOUR, OPERATIONS (*Cleidotomy*)

**Cleptomania.** See KLEPTOMANIA, INSANITY, NATURE AND SYMPTOMS (*Impulse and Obsession*)

**Clevedon.** -- See THERAPEUTICS, HEALTH RESORTS (*England*)

**Clifton.** See BALNEOLOGY (*Great Britain, Thermal and Subthermal*)

**Climacteric.**—Relating to a climacter or critical period or year in human life (Gr. κλίμαξ, a ladder), used also as a noun, climacter signifies the critical period or year itself. The multiples of seven have been considered to be specially dangerous years, such as 7, 14, 21, 28, etc., and sometimes the multiples of nine have been included. The *grand climacteric* is the sixty-third year ( $9 \times 7 = 63$ ). The term is also used as synonymous with "change of life," or the period (usually from 45 to 60) when menstruation ceases ("menopause") and the female generative organs atrophy See MENSTRUATION AND ITS DISORDERS (*The Menopause*), CLIMACTERIC INSANITY, INSANITY, NATURE AND SYMPTOMS (*Etiological Varieties*), MENOPAUSE, UTERUS, ASSOCIATED INSANITY

### Climacteric Insanity.

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See also INSANITY, NATURE AND SYMPTOMS (*Etiological Varieties*), MENOPAUSE, MENSTRUATION AND ITS DISORDERS (*The Menopause*), UTERUS, ASSOCIATED INSANITY

THE NEUROSES incident to the change of life have not been studied with that care and accuracy which the subject demands. Although it is an everyday experience in medical practice to be called upon to undertake the treatment of maladies, more or less severe and distressing, complicating the menopause, the literature of the subject is singularly scanty. Dr Tilt (first in 1853) published a book on *The Change of Life in Health and Disease*, which ran through several editions, and still remains the only English work of reference. His observations

still rank as authoritative, although his explanations of facts must be regarded as obsolete. Various studies of affections of the menopause have been made, e.g. Dr Saundby (1885) published certain observations on numbness of the extremities occurring at the climacteric, quoting Dr. Ormond and Dr Wharton Sinkler. Dr Barnes (1890) and Dr Savage (1893) have written of the neuroses of the menopause, and Dr Merson (*West Riding Asylum Reports*, 1876) has given an elaborate summary of the cases of climacteric insanity in that institution during the four years prior to publication. These communications represent a comparatively small volume of work in relation to an important epoch in the life-history of woman-kind.

It may be considered doubtful if there is a real analogous condition in men. The momentous epoch in a woman's life, when the menstrual function ceases and her general physical condition undergoes a profound alteration, is so marked and is so determined by physiological changes that it cannot fail to be taken into account, even in the most elementary consideration of epochal modifications. At puberty the child becomes a woman in form and feeling. The menstrual function is established and continues in periodic activity throughout sexual life. Any premature cessation of that function is accompanied by symptoms of ill health, requiring active medical or surgical treatment, and when, in the course of vital involution, the function is being finally obliterated, it cannot but be, especially in the complex circumstances of the artificial life of civilisation, a period of stress more or less severe. The sexual life of a man, although, no doubt, obscurely periodic in character, has no such definite characteristics. The climacteric is but feebly indicated in the process of sexual decadence. No such active revolution, intimate in its connection with mind and body, forces itself upon medical attention. The familiar facts of the gradual slowing of life's current in men are ushered in by no such profound alterations as caused Madame de Deffaud to exclaim, "Autrefois quand j'étais femme." For in this relation there is no question of premature senility, of organic degenerative nervous changes. The woman who has passed her grand climacteric is in no way enfeebled, it may be indeed that her mental powers are increased and that her physical energies are reinforced since the menstrual flux has ceased to be a recurring occasion of exhaustive and monthly disturbance. Still, when we consider the neurotic concomitants of the menopause in women, and find that these are usually marked by feelings of a melancholic type, a conscious loss of power, irritability, and general malaise, it must be admitted that similar nervous conditions do occur in men, although the period of life is

more advanced, the symptoms are more fortuitous, and recovery is less frequent.

We have the high authority of Dr. R. Barnes in stating that probably few women go through the reproductive era without some nervous disorder. They labour under painful, dangerous tension, often concealing their distress, although sometimes on the verge of breaking down.

What, then, are the symptoms of the climacteric in women? First, there is a period of unrest, of flushing, giddiness, tinnitus, headaches, and general instability. In addition to subjective signs, there are frequently floodings, or other uterine disturbance, which may pass on to organic diseases of the worst type, or may pass off and leave the person to the enjoyment of many years of excellent health. Too often these complaints are treated lightly, as part of the ordinary and inevitable routine of sexual decadence. Too often suffering is endured in silence and protracted by unnecessary reticence. But the physician is not warranted in assuming that these minor troubles preliminary to the cessation of menstruation are of slight importance and undeserving of active treatment. He can never be sure that nervous instability will not proceed to pronounced mental disorder, that floodings will not prove symptomatic of cancerous growths. Investigation and rational treatment must go hand in hand. Promptitude is essential.

Of late years the gynecologist has been frequently forced to create an artificial menopause, and much blame has been deservedly cast upon those who have been held guilty of carrying operative measures to extremes. They have been charged, in unseemly terms, with needlessly unsexing women. They have been accused of creating greater evils than they removed. Cases have been recorded where the removal of ovaries has been detrimental to mental health, and the last state of the patient rendered worse than the first. An artificial menopause has been followed by a climacteric insanity.

In the earlier stages of neurotic maladies associated with the climacteric, the bromides are in common use. I have, however, found general and considerable benefit from the administration of ovarian substance in the form of tablets. There is a disturbance of the animal economy consequent on excess of natural secretions, but there is also a disturbance dependent upon deprivation. If the sudden cessation of ovarian activity be accompanied by such symptoms as have been indicated, it is reasonable to suppose that the artificial ingestion of ovarian substance will in measure restore the balance which has been destroyed, and gradually accommodate the system to the new order of things which is imminent. It would seem that ovarian substance is in these slighter forms of nervous disturbance, at all events, a valuable remedy, to be prescribed on reasonable grounds.

Within the last few years, especially in America, few but active workers in asylums have pressed upon the attention of the medical profession their conviction of the urgent need for a more systematic, if not an universal examination of the reproductive organs of insane women. Operative interference has been advised in these cases, and one enthusiast has placed on record that, out of 100 insane women, pelvic disease existed in 93, and operative interference was required in 89. Such a statement is altogether exceptional, and has been strenuously controverted. Cases have been recorded showing that the removal of ovaries has been followed by disastrous consequences, and that these operations, generally speaking, have as little effect in the cure of insanity as the discredited operation of clitoridectomy for epilepsy. Premature cessation of function, consequent on disease or consequent on operation, an artificial climacteric, is assuredly not less hazardous than the ordinary involution of middle age.

It is well known that certain bodily diseases are accompanied by certain mental concomitants. *Sperthusa* is familiar to every physician, and the mental depression of liver disease has passed into a proverb. The influence of disorders of the sexual organs on mental states has been widely recognised. They may determine the production of hallucinations. For instance, it has been accepted that olfactory hallucinations are generally indicative of these disorders, and not less at the menopause. Even since Schroeder van der Kolk insisted on the reality of *sympathetic insanity* there has been no danger of overlooking the condition of the colon in the treatment of the insane, and perhaps more than enough has been made of the relations between the sexual and the mental apparatus. Indeed, in the active search made for underlying physical causes of insanity, the hopes which modern gynaecological treatment raised have been but partially realised. The correction of faulty conditions, however, offers a chance of mental recovery and must not be neglected.

But, it must be admitted, it must be kept in mind that the great cause of insanity is some inherited defect. Broadly, the neurotic inheritance is the foundation fact of mental disease. The stress of the climacteric period may tell severely even upon the strongest women. When that stress occurs in the case of a woman whose ancestry bears traces of mental weakness, whose mental instability is part and parcel of her very nature, the symptoms of earlier and less pressing importance, such as flushings and giddiness, should be regarded as indicative of a near possibility of further deterioration, mental and physical. The physician will seek to obviate these discomforts and disorders without loss of time, so as to prevent the development of morbid sensations into insane delusions, in so far as he can, by the regulation of vital functions and by

the details of rational therapeutics and necessary hygiene. He will regard the stress of the climacteric as a presumptive exciting cause of mental disorder in a person constitutionally predisposed to nervous instability.

At such a time a woman may very readily slip into habits of invalidism. The nervous irritability, the physical weakness, the flushings may combine to induce her to adopt a course of life which is definitely detrimental. It may be that alcoholism, offering a speedy specious relief, is an immediate danger, although it is not so common a pitfall as has been recently alleged. Alcoholic indulgence is not an efflorescence of late middle life, nor is it reasonable to suppose that the indecisive, introspective attitude so ordinarily adopted, intermingled as it is with religiosity and feelings of unworthiness, can be a favourable soil for vicious indulgence.

The question before the physician is one of redress. Equilibrium has been deranged. The habit of a generation has suddenly been broken, the system is not yet accustomed to the change of current. Manifestly his efforts must be towards the readjustment of equilibrium. This is not a mere question of medication or operative interference unless these are demanded by the very circumstances. It is rather a question of wise management of daily life, of diet, of exercise, of repose, of mental occupation and recreation than invigorating climate. To enter on the principles and details involved would mean a needless repetition of what will elsewhere be said in reference to the treatment of insanity.

**INSANITY**—It would appear that the ordinary troubles of the climacteric pass into exaggerated forms when the boundary line of sanity has been passed. By fine gradations the natural feelings of advancing years, the weakening vital impulse, the questionings of work and pleasure still remain possible, pass into realised feelings of morbid dread, of physical incapacity, of unutterable unworthiness. The flushings are exaggerated into beliefs that something has given way in the head, for continuous headache is often referred to the vertex or to the occipital region. Feelings of emptiness may alternate with the belief that some live animal is crawling about the interior of the abdomen. The skin may be the seat of delusions. It is common to find patients complain of being filthy or diseased. The occurrence of obsessions to wash is noticeable at this period of life, as it often is about the time of puberty. Naturally the genital organs participate in these disorders, or even predominate. The belief that pregnancy has occurred is very common, although this condition of *pseudo-cyesis* is by no means confined to the climacteric period, nor is it necessarily insane. Every practitioner of experience will recall cases in which insistence on pregnancy at the climacteric has caused consternation and very real trouble. Sensations have

been misinterpreted, and the cessation of the menses has been held as proof that the false interpretation of sensations has positive grounds of truth.

The varieties of insane delusions are numerous, and are developed out of these morbid feelings. As a rule, they have a sexual foundation. For instance, it is a common phase to complain of rape having been committed or having been attempted, especially in the hours of night. Not only may these patients complain of having been themselves criminally assaulted, but they may circumstantially relate that they have been made aware of friends or neighbours having been similarly maltreated. This opens up questions of medico-legal importance, which the practitioner will do well to bear in mind. He must exercise great caution in his dealings with persons so afflicted. And more especially is this to be remembered when they fall victims to epilepsy or epileptoid states, during which attacks there may be a performance of automatic actions requiring the closest observation in determining the responsibility of the parties.

The condition of life as regards marriage also determines the form of the mental malady. The old maid whose life has been narrow, and whose sympathies may have been warped, will probably suffer from an access of passionate feeling, the expiring flicker of the candle, and pester some unfortunate man with suggestions of marital relations, or accuse him of working upon her by means of electric batteries, or blowing chloroform through the key-hole in pursuit of nefarious designs. On the other hand, the married woman gives way to irritability, or dread, or disappointment, feeling that a new relationship has been established between her husband and herself. Instead of passing from the earlier years of married life to the years of a matron's duties, and so to a kindly green old age, by natural and easy transitions, the patient becomes exacting and querulous, expends her energies in a passion of jealousy, and destroys the home which she had built up with fond care. These unfortunate first steps require a world of patience and most considerate treatment. They indicate a self-consciousness and a loss of control which, unchecked, pass to easily recognisable forms of mental aberration.

THE CLINICAL FORMS of climacteric insanity are broadly resolved into melancholia and mania, with comparatively few cases of delusional insanity of a melancholic type.

Melancholia is undoubtedly by far the most common form. The cares of life, the organic malaise of the climacteric, ordinarily tinge the mental state with feelings of dread, fears of impending disasters, fears of impending disabilities. The consideration of the clinical features of melancholia need not be entered upon here. It is sufficient to correlate the

depression and delusions with the underlying physical conditions.

Mania is less common, and would be still less common were it not that alcoholism plays a certain part in the development of maniacal conditions at the climacteric. Cases occur in which melancholia follows on alcoholic excess, but the usual concomitant is mania. Again, there are cases in which there is a recurrence of mental disorder at the climacteric. These would appear to be chiefly maniacal in form.

As to the cases of delusional insanity, where the predominant features are states of fixed and limited delusions, these are but few in number, and the delusions are almost invariably of a melancholic character.

Still fewer degenerate into dementia, and these are usually complicated by alcoholic or other toxic influences.

Dr Savage has drawn attention to the observation of Dr Tilt that deafness occasionally occurs at the menopause—a defect which may be temporary or permanent. No doubt tinnitus is frequently a symptom of the climacteric, and it may pass on to more serious conditions, but it does not appear that deafness is in any way peculiar in its mental complications at that particular period of life. It is a matter of common observation that deaf persons often become suspicious, morose, delusional, and dangerous. Those afflicted with congenital or acquired deafness and insanity are among the most dangerous of the inmates of asylums. They are nearly always persecuted and persecutors.

In addition to the mental aberrations above described and grouped according to preponderating symptoms, allusion must be made to certain moral aberrations which come under the notice of the physician from time to time. Reference has been made to the alcoholic habit which may begin with the troubles of the menopause, but which is more probably only a shameless exacerbation at that period. The excuse comes so readily that it gives the impression of having been too easily produced, just as the excuse may have been dysmenorrhœa or puerperal troubles in earlier life. It is not usual to find that a woman of alcoholic habits gives up her vicious indulgence on the cessation of climacteric disorders, on the contrary, it would appear that she continues to get drunk because she dislikes being sober. The deep-seated untruthfulness of the drunkard must be discounted in arriving at any opinion on this question. And, similarly, it is not in accordance with modern experience to expect that mental disease of long standing will cease and determine on the completion of the menopause.

More serious, if possible, are those cases of moral insanity in which nymphomania becomes a prominent symptom. Fortunately these are rare, but when they do occur they are only too easily recognised.



**Prognosis.**—Having thus examined the general facts relative to mental disorders occurring at the climacteric, it is evident that there are no marked peculiarities other than can be readily understood on consideration of the underlying physical and environmental conditions. It will consequently be granted that on the cessation of the period of stress there is expectation that the recoveries will not be fewer than in connection with other similar disorders. Indeed, it may be averred that the chances of recovery are more than usually favourable, if we exclude cases complicated by previous attack, organic cerebral lesions, and the manifestly degenerate. Still recovery may be protracted, though but few die. The risk of death by suicide is comparatively small, notwithstanding the prevalence of gloomy thoughts and feelings of unworthiness, for the slower current of life enfeebles the power to end misery by self-destruction. Yet while this is generally true, it would be a reckless procedure which would fail to take such a risk into account and fail to take precautions for prevention.

The treatment of climacteric insanity must proceed on the theory of individual necessities. If the sudden cessation of ovarian activity is causing the mental instability, as has been already indicated, ovarian substance promises good results. If the symptoms are referable to the skin, and a harsh dryness indicates sluggishness of action, baths and exercise will be indicated. There is no class of cases for whom exercise is more generally beneficial, but exhaustion may be so profound that rest and massage are preferable. Or if the alterations in the appendages of the skin are the basis of delusions, these must be treated. Removal of unsightly hair from the face has led to recovery. Again, there is often constipation of an intractable form, leading to autotoxic effects. There is no better remedy for this than sulphate of magnesia in small and daily doses. These indications of treatment will serve to show how closely the physical condition must be studied, and how treatment must be appropriate to that condition. It may be that the gynecologist will be required to operate upon new growths or to rectify malpositions. In short, if the treatment of insanity is to be successful, there must be no omissions in research and no failure in performance. This is not the place to enlarge upon those measures of mental hygiene which such patients require, nor is there any special note of interest in reference to the place of treatment. If appropriate treatment can be had without having recourse to asylums, no doubt that will be tried, but it will be recognised that, for the vast majority of cases of pronounced climacteric insanity, the special appliances and practical experience at the service of the public in a modern asylum are both requisite and necessary.

VOL. II

## Climate and Acclimatisation.

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See also DIET (*Modifying Factors, Climate*), LIVER, TROPICAL AFFECTIONS (*Hyperæmia, Effect of a Tropical Climate*), LUNG, TUBERCULOSIS OF (*Treatment, Therapeutic, Climate*), MENSTRUATION AND ITS DISORDERS (*Puberty, Age of*), METEOROLOGY, NEPHRITIS (*Etiology, Climate*), OBESITY (*Etiology, Race and Climate*), SKIN DISEASES OF THE TROPICS (*Caused by Climatic Conditions*), THERAPEUTICS, HEALTH RESORTS (*Climate and its Effects*)

CLIMATE, from the Greek word κλίμα, I incline. Lat *Clima*, Gr κλίμα, a region, Fr *Climat*, Ger *Edtstrich, Himmelstreich*

The word climate is now used to specify the definite differences which obtain between one region and another, as regards temperature and its variations, humidity and rainfall, the composition of the atmosphere, its density and pressure, winds, electrical conditions of the atmosphere, the organic and inorganic substances it contains, etc. The configuration of the ground must also be taken into account, the composition of the soil and the presence or absence of vegetation, marsh-land or desert, plain or valley, inland or maritime position, mountainous or otherwise.

The term used to be employed in astronomical or mathematical geography to designate "a portion or zone of the earth's surface, comprised between two lines parallel to the equator, and measured by the length of time during which the sun there appears during the summer solstice, that is, by the sun's inclination. The space between the equator and the pole was divided into half-hour climates, in which the length of each day increased by half an hour, and also into monthly climates."

As the climate of a region may be said to be essentially dependent upon the duration of its exposure to the sun's rays, modified certainly by local conditions, the relation of the region to the equator is of great importance. Hence, originally, three great climatic divisions were described—

1 The hot or warm climate, extending from the equator to lat 35°, with a mean annual temperature of about 80° F (27° C)

2 The temperate climate, extending from the 35th to the 55th degrees of latitude, with a mean annual temperature of about 60° F. (16° C)

3 The cold climate, between 55° lat. N and the poles. In this region the temperature varies from 40° F (5° C) to 5° F (–15° C)

A more scientific division of climates is made by using isothermal lines, because the mean annual temperature varies in different regions occupying the same latitude on the earth's surface. This would give us five fairly extensive regions between the equator and either pole—

1. The hot region, between the equator and the isothermic line of 77° F (25° C)
2. The warm region, between the isothermic lines 77° F (25° C) and 59° F (15° C)
3. The temperate region, between the isothermic lines 59° F (15° C) and 41° F (5° C)
4. The cold region, between the isothermic lines 41° F (5° C) and 23° F (-5° C)
5. The polar region, between the isothermic lines 23° F (-5° C) and 5° F (-15° C)

Apart from temperature, rainfall, winds, etc., to which reference will be made presently, for practical purposes we must refer to seven climates—

- 1 Hot climate
- 2 Temperate climate
- 3 Cold climate
- 4 Insular climate
- 5 Maritime climate.
- 6 Continental climate
- 7 Mountaneous climate

As all these climates have general differences, it will be well to briefly describe them before referring to details

**GENERAL CHARACTERISTICS**—1 *The hot climate*, which may be described as practically extending to 35° north and south of the equator, contains within its limits South Asia, nearly all the islands of Polynesia, the greater part of Africa and its islands, and those parts of North and South America lying between the latitudes mentioned

In these regions the heat is greatest, not, as might be imagined, at the equator, but at the tropics (20°-23°-30°). The heat at the equator is modified by the equatorial rains, which fall almost daily, and the equatorial calms, but although the rainfall is fairly equally distributed during the whole year it only amounts to about 45 inches in this equatorial region. Between 5° and 10° of latitude there are two rainy seasons in the year. In regions between 10° and 13° there is one rainy season, which lasts upon an average for five months. These general considerations are modified by the monsoons

The variation in the thermometer is slight during the day, but at night there is often a considerable fall. In general terms, the range of temperature is from about 55° to 120° F (13° to 49° C). The daily variation in barometrical pressure is well marked, but the general variation is slight

There is an almost rainless area north and south of the equator from about 16° to 28°, where it seldom or never rains, and in these belts the mean annual temperature is greatest

Throughout the hot climates thunderstorms are of frequent occurrence

The general influence of the hot climate upon the inhabitants is to render them lazy and apathetic. It has a very definite influence upon the constitution of Europeans residing there, and the physiological functions of the body are distinctly affected. With regard to the normal temperature of the body, we find that, according to numerous observers, it is slightly raised, probably about 0° 40 F (0° 04 C), but the mean diurnal difference of temperature is practically the same in Europeans as seen in Europe. Crombie found that the mean temperature of Europeans in Bengal, taken in the mouth, was 98° 49, with a maximum daily range of 1° 31, as compared with the English average of 98° 084 and 1° 41 respectively. With regard to respiration, Rattray's observations are summarised by Davidson thus: "The vascularity of the adult lungs is reduced by 12 to 13 fl oz, their spirometric measurement by the average of 32 inches, their function by 18 43 per cent, that is, they use 36 85 cubic feet less of air daily, the excretion by 1 84 oz less of carbon and 6 7 loss of watery vapour." This is accounted for by the number of respirations being lessened to about 14 per minute, and there being considerably less blood in the lungs under the influence of an average temperature of from 80° to 83° F (27°-28 5° C).

There is a very slight difference in the pulse rate, and the proportion between the respirations and the pulse rate is not the same as in Europe. The amount of urine voided is diminished in quantity. Rattray says that it is diminished by 17½ per cent, but, of course, the amount of urine depends upon the amount of fluid consumed, the temperature, and the humidity of the atmosphere

Opinions vary as to the effect of a hot climate upon the blood, and for this reason that probably malaria must be taken into consideration. On the whole, however, it is safe to say that Europeans suffer from a slight anemia. By some authors this is considered physiological anemia, and rather beneficial than otherwise

The nervous system during the first few months of a residence in a hot climate is excited, but this excitement soon gives rise to depression, which latter is partly due to loss of sleep, partly to climate, and also to the worries incidental to a residence in a hot climate

Digestion is slower than it is in Europe, and digestive powers are weakened, the appetite fails, and nutrition is diminished, weight is lost, and the muscular system enfeebled. The functions of the liver and skin are greatly stimulated. Menstruation commences about one or two years sooner than in Europe

European children born in hot climates thrive fairly well in infancy, apart from their liability to suffer from diseases incidental to the climate.

The *mortality* in the hot climates in new arrivals is chiefly due to fever, heat apoplexy, and intestinal disease, but after a year or two's residence these diseases show a marked diminution in amount. It must also be admitted that sanitary measures and a more careful mode of living have greatly reduced the amount both of disease and mortality in Europeans in hot climates, thus the death-rate from disease in India among soldiers, excluding cholera, has decreased by two-thirds since the 'sixties, when the British soldier in Bengal died at the rate of some 60 per thousand. The death-rate of soldiers in Bengal in 1888, excluding cholera, was 10.54 per thousand. The mortality of the women, however, was somewhat higher. The death-rate of soldiers' children under one year was 189.64 in 1888, but it must be remembered that in India they do not suffer from the want, privation, and exposure to which children are subjected in the large towns of England, where the death-rate is very little lower, and in order to estimate these figures aright we must also remember that, as Davidson points out, "the diminished mortality is the result of withdrawing soldiers' children from the tropical influences, which proved so injurious, and rearing them in a temperate climate", this is done by taking the children from the plains to establishments in the hills in the hot season.

2 *The Temperate Climate*—This climate is the healthiest upon our globe, as neither the extremes of heat nor cold are experienced. It may be said to be situated between latitudes 35° and 55°. The mean annual temperature varies from 50° to 60° F (10°-16° C). In the temperate regions we have the four seasons—spring, summer, autumn, and winter,—and there are probably greater differences in local climates than elsewhere. It is in this area, too, that the influence of large towns and country districts must be taken into account, for, undoubtedly, the overcrowding which obtains in great cities does not tend either to longevity or to robust health, whilst infant mortality is very high. Great differences, however, obtain in different places, owing to the advance which sanitation has made. If we compare the death-rates of the various capitals of Europe we find marked differences, for instance, the death-rate in London is one-third less than in St Petersburg or Moscow. Certainly the healthiest climates in the world are to be found in this temperate zone, and we possess far more accurate knowledge with regard to the diseases of this area than of any other. As will be seen in the article on "Health Resorts," they are practically all to be found in temperate regions.

3 *The Cold Climate*—This climate, which covers the areas from about 55° F. (13° C) to the pole, has been divided by some observers into three—the cold, having a mean annual temperature of from 40° to 50° F (5°-10° C),

the very cold, a mean annual temperature of 32° to 40° F (0°-5° C.), and the glacial, where the mercury is below freezing-point. In the northern hemisphere, northern Russia, Lapland, Finland, Iceland, Norway, Sweden, Denmark, the north of Scotland, Spitzbergen and Nova Zembla, Canada, northern Asia, Siberia, and Kamtschatka, as well as Greenland, lie within it.

Although cold severely tries the lungs and kidneys, yet, according to M Levy, the death-rate in this region is the lowest in the world. His estimate with regard to it may here be quoted—

From 0°-20° latitude, 1 death takes place in 25 inhabitants, from 20°-40°, 1 in 35, from 40°-60°, 1 in 43, from 60°-80°, 1 in 55.

The inhabitants of this region are vigorous and muscular, their powers of digestion are remarkable, but their nervous systems are not highly developed.

4 *The Insular Climate*—This climate is remarkable in many ways, the seasons are more equable, owing to the surrounding water. The air is constantly changed by the prevalence of winds, and is charged with abundance of moisture.

5 *The Maritime Climate*—This much resembles the insular climate. It is warmer in winter and cooler in summer than the continental climate, for, as a rule, the range of temperature increases from the coast towards the interior. The annual range of temperature on the coasts of the great oceans is diminished to about 20° F (-7° C), whereas in the interior of a large continent, as, for instance, in the centre and north of Asia, the range may be from 60° to 100° F (16°-38° C).

6 *The Continental Climate*—This climate has a tendency to extremes of temperature—cold winters, hot summers,—and even at a short distance from the sea these changes obtain.

7 *The Mountainous Climate*—Here we have a low barometric pressure due to altitude, the air being more and more rarefied as we ascend. The heat diminishes, but the mountains attract clouds and watery vapour, thus notwithstanding, the climate is unusually healthy. The air is exceptionally pure. On first going to high altitudes, the respirations and pulse are accelerated, and the amount of carbonic acid and water exhaled by the lungs is increased, but after a residence of from one to four weeks, the pulse falls to normal, the respiration, however, continuing fuller than it was on the plains.

A consideration of the ocean climate will fall more naturally under the heading of "Health Resorts," but it may be said to be essentially characterised by warmth, equability, and excessive moisture.

INDIVIDUAL FACTORS—Considering now more in detail, but yet briefly, the various factors which are summed up in the comprehensive

term of climate, we turn first to the *composition of the air*. Roughly speaking, in 100 volumes of air, we find there are of oxygen 20.96, nitrogen 79.00, carbonic acid 0.04. The amount of oxygen in the air varies to a considerable extent, but rarely exceeds one-tenth in volume, the carbonic acid in the atmosphere varies considerably also; it may be in the proportion of from 4 to 30 in 10,000, there are also in the atmosphere other substances of a gaseous nature—ammonia, ozone, etc., as well as solid substances floating in the air, which vary in different places and under various conditions. Obviously in large towns one would expect these adventitious particles to be far more abundant than in the country. The suspended matters in the atmosphere are of considerable importance, and indeed it is upon the recognition of these that Lister's antiseptic theory has been based.

The *temperature of the air* naturally also influences climate, and in this connection we have to distinguish between the sun heat or radiant heat, and the air heat or shade temperature, which is due to the warmth imparted to the air from the ground, unless influenced by wind. "The nature of the surface of the ground exercises great influence on the amount of heat which is absorbed and reflected, the nearer the colour of the ground approaches to white (snow, chalk cliffs, white walls, etc.), the more direct the sun heat reflected by it, the less heat being absorbed, the darker the ground (grass, green leaves), the less heat reflected and the more absorbed. The ground which absorbs more heat from the direct rays of the sun can give out more heat during the night, and *vice versa*. The influence thus exercised on the climate of a place is evident, and the white snow-fields of the Alps in winter form a well-known illustration."

The temperature of maritime regions is influenced by the ocean, and in mountainous regions altitude lowers the mean annual range of temperature.

With regard to *barometric pressure*, it must be noticed that, if great, it exerts a very marked influence upon the human being. It tends to increase the amount of carbonic acid and water exhaled by the lungs, and at first to induce laboured respiration and inability for exercise, as well as constipation and depression.

The *influence of light* is also important, and, owing to its action on the centripetal nerves, it increases the metabolism.

*Winds* purify the air and induce changes in temperature, atmospheric pressure, moisture, and light, and therefore they must be taken into consideration, not only with regard to climate in general, but as to health resorts in particular. The monsoon winds, the sirocco, and the kamisin are beneficial or untoward factors where they obtain.

The *cultivation of the soil* exerts an influence upon the temperature, for cultivated land, which is well drained, raises the temperature, as has been demonstrated by Buchan. It is obvious also that vegetation must play a marked part, because where it is luxuriant the sun's rays upon the ground are more or less prevented. Ground covered by forests has a lower temperature than that which is bare, and the diurnal variation in well-wooded districts is more equable than that in open country. It is obvious also that sandy, dry, and well-drained soils are much warmer than wet and marshy districts, which certainly produce mist and fog. A sandy desert is exceptionally warm in summer, although comparatively cold in winter.

The amount of *water vapour* in the atmosphere is also an important factor of climate. Evaporation and condensation constantly alter the humidity of the atmosphere. As a rule, absolute humidity is greatest with a high temperature, but relative humidity is usually greater in winter than in summer. It is only when saturation of the air obtains that we have mists and clouds,—clouds predominating at average heights, mists clinging to plains and maritime positions, or the neighbourhoods of large lakes. On mountain tops mists are rare.

With regard to *rainfall*, its distribution is unequal over the globe and varies in amount, from 300 to 400 inches annually on the southern slopes of the Himalayas, to almost nothing in the Sahara. Again, the number of rainy days experienced annually varies greatly, and does not invariably correspond to the amount of rain which falls. Rainfall is not always injurious, as it diminishes the amount of impurity in the atmosphere, but, in considering the climate of a health resort, the amount of rain, the rainy season, the number of days on which rain falls, and the time of the day, are all subjects for consideration, as far as invalids are concerned.

It should be remembered that where mountainous regions lie in the path of moist air currents rainfall is great, as, for instance, the Khasi range of hills, which have an annual rainfall of about 500 or 600 inches, for they intercept the S.W. monsoon, which, laden with vapour from the Bay of Bengal, impinges on them. On the lee side of mountains, however, the rainfall is less.

Although the electrical condition of the atmosphere must play some part in climatology, its practical importance is insufficiently understood.

Climate must also be considered with reference to the treatment of disease. A change of climate cannot be said to be a specific in treating disease, but there is no doubt that in many diseases a change of climate acts most efficaciously. It is a well-known fact that the removal of a patient for even twenty or thirty miles may be exceedingly beneficial. In choosing a change of

climate for an individual, the patient's personal equation or idiosyncrasy must be taken into account. In dealing with patients suffering from nervous diseases, in whom we wish to restore the general tone of the system, we should choose bracing and moderately temperate climates, remembering that proximity to the sea may induce nervous excitement and insomnia, and may, temporarily at least, exaggerate neuralgias. Diseases of the lungs, apart from phthisis, are favourably influenced by a climate characterised by moisture and mildness. A bracing equable climate is requisite in cardiac disorders, remembering that pulmonary complications due to climate should be avoided. A moderate altitude is usually beneficial, but the patient must not be sent too rapidly to a high altitude, and very sudden changes of temperature must be avoided. In diseases of the abdominal organs, such as intestinal catarrh, chronic liver disease, dyspepsia, dysentery, diarrhoea, etc., change of climate may be most beneficial, climates such as the Riviera, the Nile, St. Montz, and Morocco being advisable. This will be further considered under "Therapeutics, Health Resorts."

**ACCLIMATION.**—The question of acclimatisation naturally falls under the subject of climate. In recent years two schools of thought have been formed—one regarding acclimatisation as impossible, a view supported by Virchow, Hirsch, Fritsch, Ravenstein, and others, who hold that Europeans can never become acclimatised in the full sense of the word in tropical regions, believing that the deterioration caused by climatological factors and endemic diseases will invariably kill off the emigrants, or at any rate render their ultimate existence impossible, the other school, represented by De Quatrefages, Livingstone, the late Bishop Hannington, and more recently by Dr. L. Sambon, believing that rapid acclimatisation in tropical regions is possible for Europeans. The writer of this article holds that rapid acclimatisation in tropical regions is impossible, and that acclimatisation for Europeans can only be possible if migration occur step by step. In estimating the possibilities of acclimatisation we must count by generations rather than by years, although, given carefully selected individuals and carefully selected tropical areas in which to colonise, he sees no reason why, with precautions, Europeans should not colonise even in the tropics. In making the selection, all persons with a tendency to gout or rheumatism, diabetes or albuminuria, those with a nervous or alcoholic family history, or those suffering either from acquired or hereditary syphilis should certainly be rejected.

Acclimatisation is a process, usually slow, by which plants and animals become adapted to, and so retain health in, countries having a different climate from those in which they are indigenous, it is in part effected by changes taking

place in the individual or in the race, in part by hereditary modification of constitution. If we look at the distribution of different races throughout the world, we find that great changes have taken place in their location, the Esquimaux once lived in Asia, only some 40° north of the equator, now they inhabit the polar regions, the Bohemians or Gipsies are found nearly everywhere, while Jews and Maltese apparently thrive in every conceivable region. But, examining the matter more closely, we find that the Esquimaux did not arrive at their present hunting-grounds in a generation, much less in a few weeks, as emigrants and soldiers are compelled to do now, nor do we find that the Gipsies or Jews overspread the world rapidly. Far from it, they put out feelers, as it were, and only very gradually, year after year, generation after generation, did they advance from their native soil into the great unknown. Looking for a moment at other races, we observe that the British become acclimatised at the Cape, in Southern Australia, in New Zealand, and in some parts of North America, but not in India. The French thrive in Nova Scotia and in Mauritius, but not so well in the north of Algeria, although in the southern provinces they do better. The Dutch fare exceedingly well at the Cape, but not in the Malay Peninsula, where they have experienced fearful mortality. Madagascar may be instanced as a place having a climate which has proved most unfavourable to all Europeans.

In dealing with the subject of acclimatisation with reference to Europeans, an error is made in imagining that all Europeans can be acclimatised or will resist acclimatisation in any given area equally, not so, a marked difference obtains between northern and southern Europeans in this respect. Not only the climatology of their original residence must be taken into account, but also their habits and customs and their psychological peculiarities, and if these factors have to be considered with regard to the adaptability of a nation for emigration, so too with regard to the individuals of any nation. Selected individuals from nearly every European nation may thrive almost all over the world. Some have denied that environment influences the human species, and it has been said that nations seek out that environment which is best suited for them, but environment certainly does definitely influence not only individuals but nations. Herbert Spencer has shown in his *Principles of Biology* that every organ and every function of living beings undergoes a certain and definite modification, within certain limits, under the stimulus of new conditions, and he thinks that this modification is almost always such as to produce an adaptation to a new environment.

The influence which climate and environment exert upon emigrants is well shown in Australia, New Zealand, and America, proving that a race

such as the Anglo-Saxon may undergo material changes. It is obvious that the Colonials of the present day are practically different from their ancestors, and they indicate a tendency to change to an altogether new type of manhood, with new aptitudes and capabilities.

Broadly speaking, Europeans can only become rapidly and readily acclimatised in the temperate zone, that is to say, where climatic and other conditions are approximately akin to their present habitat. It may also be said that people who inhabit the temperate zone become more easily acclimatised in countries towards the north of their present habitation.

It is interesting to notice that the peoples of Southern Europe, such as the Italians and southern Frenchmen, can better bear the climate of sub-tropical Africa than can Northern Europeans, and, as Mr Ravenstein has pointed out, "a steady stream of migration is, in fact, setting in that direction. Germans and Belgians are pouring into France, Frenchmen are going to Algeria, the Arabs from the shores of the Mediterranean have found their way into the Sudan, whilst the Sudanese are pushing forward into Bantu Africa. The descendants of those Dutchmen who, a couple of hundred years ago, first settled at the Cape, have made their way to the Transvaal, and European migration, favoured by geographical features, is being pushed, even within the Tropics, towards the Zambesi."

In treating the subject of acclimatisation, some have simply divided mankind into two classes, white and black, but this is a far too sweeping generalisation, and it must be examined more closely if a definite and right conclusion is to be arrived at. An endeavour must be made to prove with certainty in each separate case what power of resistance is possessed by any given national constitution, in order to decide whether it may successfully acclimatise itself in a new country and permanently colonise. In white races there is apparently a marked gradation in their susceptibility to climatic influences, and a very decided difference is noticed between the Aryan and Semitic races in this respect. The Arabs and the Jews are both unequalled in their power of adapting themselves to new environment, but even between them a difference exists, in so far as the Jews appear to continue definitely capable of reproduction, although they marry amongst themselves, while the Arabs often suffer from degeneration which is only retarded by the introduction of new blood. They take wives from the races among whom they settle and thereby continue to exist, but this is not true acclimatisation, for the race is altered and transformed into a mixed race, which eventually possesses very little in common with the original stock. Race itself does not always provide us with a definite clue to capability of acclimatisation, for in India the Hindu popula-

tion, notwithstanding its Aryan origin, has thriven under unfavourable circumstances and even in malarial districts, presenting a striking contrast to the English (also an Aryan race), whose intolerance of the Indian climate is obvious. Temperament also plays a not unimportant part in the possibility of prolonged existence in a given region. It is a quality which requires considerable time to be modified by new conditions of life, e.g. the Indian at Brazil is dull and sullen, the negro is vivacious and gay, although in the same climate. Hereditary temperament and capacity of mind characterise variously all races, and differences of intellectual and moral power are well-marked factors in the possibility of acclimatisation. Virchow not long ago pointed out, with reference to acclimatisation, that two distinct questions are usually confused, viz. (1) How long can any single individual, with precautions and care, live in any particular climate? (2) what races can thrive and colonise in any particular climate? No definite answers are yet possible. In answer to the first it may, however, be said that selected individuals may live for a time anywhere with precautions. With regard to the second, rapid acclimatisation of a race can only be obtained if it migrates to regions having approximately the same climatic conditions as its original habitat. If Europeans, for instance, attempt to colonise in the tropics, they almost invariably die out. In the West Indies we have a striking exemplification of this, and even in Cuba, which is brought forward by some authorities as a proof to the contrary, the apparent increase in the white population is more than accounted for by immigration. The permanent decrease of the white population in the West Indies dates from the abolition of slavery, as overseers and occupiers of the soil, Europeans are able to withstand climate more successfully than when they are compelled to undertake actual manual labour. The white man's incapacity for manual labour is an important question throughout the tropics, it has necessitated the employment of Chinese labour in America, of coolies in Mauritius and Java, and of kidnapped natives from the South Sea Islands in Australia.

From what has been said it is obvious that Europeans are *almost* incapable of colonising in the tropics—*almost*, because there are some places in the tropics occupying a high altitude, and some islands whose climate is so modified by the surrounding water and by their exposure to winds, that they almost possess a temperate climate. For instance, in the Vindhya hills in India the French have colonised successfully, and, theoretically, there is no reason why Europeans should not colonise and thrive in some of the highlands of Central Africa, when railway communication has been provided to carry them rapidly across the dangerous belt of malaria on the coast.

If we desire an example of a race thriving in extreme vicissitudes of climate, it is to be found in the Indians, who, coming from a temperate region in North America, have become acclimatised in the hot dry coasts of Peru, and also in the extremely cold regions of the Andes at an altitude of 4000 to 6000 feet. Now, notwithstanding that the inhabitants of these two regions will not thrive if they are removed from one to the other, they have nevertheless proved the possibility of an original race becoming gradually acclimatised in areas which are totally different from a climatological point of view.

Bertillon well summed up the circumstances which go far to prevent sudden acclimatisation in new isothermal regions —

1. Acute diseases, many of them endemic.

2. Chronic consecutive anemias, which place the individual in an unfavourable position to resist accidental disease.

3. Diseases in early infancy in offspring in the new home.

4. Physical and intellectual degeneration and the infertility of the second and third generations.

That climate markedly affects the progeny of emigrants is certain, for instance, when Europeans proceed to countries where they can colonise, which possess a very different climate from their own, their children in a generation or two receive an indubitable impress from the climatological factors around them. The children of Europeans who go to North America tend to approach in type to the original inhabitants of the soil, they outwardly resemble the north-west American Indians, the hair becomes straighter, coarser, darker, the cheek bones more prominent, and the rounded form of the face changes into the gaunt-lined face typical of the inhabitants of the United States, the skin becomes somewhat sallow, and even the voice changes. Again, supposing a woman proceeds to Australia, having given birth to three or four children, the children she subsequently bears in the new climate will not resemble her previous progeny so much as they do the Australian type. In tropical countries we find that the children of northern European families do not thrive, and the race dies out, not so much because of the infertility of the women, but because of their inability to bear children capable of thriving. In those cases where the women are sterile it is due to the action of the climate upon them as Europeans, and not to any specific action on the sex.

**Climatology.**—The study of climates and of the effect they produce on the health of the individual or of communities. See CLIMATE.

**Cliniatria.**—Clinical medicine, is derived from *κλίνη*, a bed, and *ιατρεια*, treatment.

**Clinic or Clinique.**—A gathering of medical students or post-graduates in a

hospital for the purpose of acquiring a knowledge of clinical (or bedside) medicine or surgery, sometimes the name is given to the patients (clinical material) gathered together for teaching purposes, or even to the building in which the instruction is given.

**Clinical.**—Relating to practical or bedside treatment or instruction in medicine or surgery, as opposed to theoretical lectures and demonstrations, e.g. Clinical Medicine, Clinical Surgery, Clinical Gynaecology, etc. See ABDOMEN, CLINICAL INVESTIGATION, CHILDREN, CLINICAL EXAMINATION OF, GYNECOLOGY, DIAGNOSIS IN, etc.

**Cli-no-cephaly.**—A malformation of the head in which it is flattened on the top, saddle-shaped, it is due to synostosis of the parietals with the sphenoidal great wings or with the squamous temporals.

**Clinodactylism.**—The malformation of the foot or hand in which one digit overrides another.

**Clinoscope.**—An instrument for detecting and measuring deviation or declination in ophthalmology.

**Clitoris.**—The erectile organ situated at the apex of the vestibule in the anterior region of the vulva. See GENFRATION, FEMALE ORGANS OF (*External*). It has been removed (*clitoridectomy*), but with very doubtful benefit, in cases of nymphomania. See also LABOUR, POST-PARIUM HÆMORRHAGE (*Primary, Traumatic*), SYPHILIS (*Primary*), TAKES DORSALIS (*Symptomatology, Genital Organs*), UTERUS, MALFORMATIONS OF (*Hypertrophy of Clitoris*), VULVA, DISEASES OF THE (*Tumors, Morbid Conditions of Clitoris*).

**Cloaca.**—The space or opening into which the intestinal and uino-genital canals discharge, a cloaca exists normally during embryonic life in the human subject, and it may remain permanently as a malformation (see GENERATION, FEMALE ORGANS OF, *Malformations*), or be produced traumatically during a prolonged or instrumental labour (PELVIS, PERINEUM AND PELVIC FLOOR, *Lesions*), the name *cloaca* is also given (in surgery) to cavities containing pus, or to holes in the involucrem of new bone in diseases of bones, e.g. in acute osteomyelitis and periostitis (see BONE, DISEASES OF).

**Clonic.**—*Clonic* (from *κλόνος*, irregular motion), as applied to movements, signifies irregularly occurring contractions alternating with relaxations, e.g. in epilepsy, eclampsia, and hysteria, it is opposed in meaning to *tonic*.

**Clonus.**—A series of contractions of the muscles of a limb or part of the body, causing rhythmical jerks or movements, e.g. ankle clonus,

wrist clonus, knee clonus (patellar reflex), produced in normal or abnormal amount by tapping or sharply stretching one of the tendons. See TENDON-JERK.

**Cloquet's Hernia.** See HERNIA, FEMORAL (Pectineal Hernia)

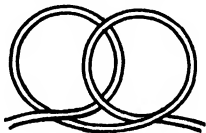
**Closet.** See SEWAGE AND DRAINAGE

**Clothing.** See DISINFECTION (Practical), NURSERY HYGIENE (Clothing), PREGNANCY, MANAGEMENT (Clothing)

**Clouds.** See METEOROLOGY (Fog and Mist)

**Cloudy Swelling.**—Swelling with turbidity (in excess of the normal) of the protoplasm of the cellular elements of a tissue, due to interference of some sort with the life of the cell or its metabolism, the turbidity is due to the presence of granules in the cytoplasm, and the granules are either extraneous substances taken into the cell or precipitated matters which are normally present but in solution, the swelling is caused by imbibition of water, fatty degeneration may occur as a subsequent change or the cells may return to the normal, the organs whose cellular constituents are thus affected (kidneys, liver, heart) are larger in size, paler in colour, and of a softer consistence than normal. Among the common causes of cloudy swelling are toxins (e.g. of fevers), heat, and certain organic and inorganic poisons (e.g. abrin, corrosive sublimate)

**Clove-Hitch.**—A catch or noose for temporarily fastening things together or for making traction on a part, in surgery a bandage or towel may be used for this purpose, the bandage so used is passed twice round the limb in such a way that both ends pass under the centre part of the loop in front, thus—



**Cloves.** See CAROPHYLLUM

**Clownism.**—The stage in a hysterо-epileptic fit during which curious contortions occur along with marked signs of emotion, it is preceded by epileptic or epileptoid convulsions, and is usually followed by the assumption of an attitude expressing fear or ecstacy

**Club-Foot.** See DEFORMITIES (Lower Extremity, Foot), ANKLE-JOINT, REGION OF, DISEASES (Tubercle Arthropathy), BURNÆ, INJURIES AND DISEASES (Simple Chronic Bursitis), HYDROCEPHALUS (Complications)

**Club-Hand.** See DEFORMITIES (Hand, Club-Hand).

**Clubbing of Fingers and Toes.** See BRONCHI, BRONCHIECTASIS (Clinical Phenomena), LUNG, TUBERCULOSIS (Complications, Integumentary)

**Clupea Thyssa.**—A poisonous fish, the sardine doré of the West Indies See SNAKE-BITES AND POISONOUS FISHES

**Clyster.**—A clyster (Gr κλύειν, to wash out) is an enema or injection, used for the purpose of administering nutriment per rectum, or for procuring an evacuation of the bowels, the name is also applied to the pipe, syringe, or tube which is used

**Cnemial.**—Relating to the leg or tibia, from Gr κνήμη, the leg, thus *cnemelephantiasis* is elephantiasis affecting the leg, *cnemitis* is inflammation of the tibia, *cnemolordosis* is forward curving of the leg, and *cnemomolosis* is lateral curving of the leg

**Cnesis.**—Itching The word is derived from Gr κνίω, I scrape or scratch, and *κνισμα*, an itching, thus *cnematomorpholyc* is itching pomphigus, and *cnethocampa* the name of a species of caterpillar which causes urticaria epidemica in some parts of Switzerland

**Cnidosis.**—Urtication or the sensation of being stung by a nettle (Gr κνίδη, a nettle), the skin disease, urticaria

**Coagulation.**—The conversion of a fluid (the blood) into a solid jelly, followed in a short time by the separation of the clot from the serum See BLOOD (Plasma and Serum, Nature of Coagulation), PHYSIOLOGY, THE BLOOD (Clotting or Coagulation)

**Coagulation-Necrosis.**—A secondary post-necrotic change in the tissues, characterised by swelling and transformation into "homogeneous masses of an increased consistence," as seen in infarcts of the kidney and in other tissues in toxic states

**Coagulins.** See BLOOD, TEST FOR HUMAN, PRECIPITINS

**Coal Gas.**—The gas obtained by the destructive distillation of coal, consisting of olefines (ethylene,  $C_2H_4$ , methane,  $CH_4$ , etc), hydrogen, carbon monoxide, carbon dioxide, nitrogen, and sulphur compounds (the three last-named constituents being impurities) See VENTILATION AND WARMING Water gas, which is produced by the action of superheated steam on red-hot fuel, consists of carbon monoxide and hydrogen, with hydrocarbons or illuminants added to it (carburetted), it is poisonous and ought therefore to be "odorised," for it has no



smell. See TOXICOLOGY (*Gaseous Poisons, Carbon Monoxide*).

**Coal-Miner's Dermatitis.** See DERMATITIS IN COAL-MINERS

**Coal-Miner's Lung.** See ANTHRACOSIS, LUNGS, PNEUMONOKONIOSIS

**Coaline.**—An alkaloid obtained from decomposing meat See INTERTINES, DISEASES OF (*Enteritis, Exciting Causes*)

**Coaptation.**—The careful and exact approximation of two separated parts (the ends of the bones at a fracture) to each other

**Coarctation.**—The constriction or diminution in size or calibre of a part, *eg* a blood-vessel or the intestinal canal

**Coat.**—A layer of tissue, a membrane, or covering (*eg* muscular and mucous coats) See also BUFFY COAT

**Cobalt.**—A metallic element (Co), occurring in nature as the arsenide (CoAs<sub>2</sub>), or as the arsenide and sulphide (CoAsS, or cobalt-glance), solutions of cobalt chloride form a *sympathetic ink*, the salts of cobalt are poisonous and kill by arresting the heart's action, primary cancer of the lung is remarkably common in the cobalt miners of Schneeberg

**Cobras.** See SNAKE-BITES (*Elaapida, Naja*)

**Coca.**—Coca or Cuca consists of the dried leaves (*Coca Polva*) of *Erythroxylon Coca*, it contains the alkaloids cocaine (methylbenzoyl-ecgonine), cocaineum (isatrophyl-cocaine), and cinnamyl-cocaine, it has an official preparation, the *Extractum Coca Liquidum*, given in doses of  $\frac{1}{2}$  to 1 fl dr, and its action is due to the cocaine contained in it See COCAINE

**Cocaina.** See also COCA, COCAINE—*Cocaina* is insoluble in water, but soluble in alcohol, ether, chloroform, and olive oil *Preparation*—Unguentum Cocaine, strength 4 per cent *Cocaine Hydrochloridum* is a crystalline powder, freely soluble in water, alcohol, and glycerine *Dose*— $\frac{1}{2}$ – $\frac{1}{4}$  gr *Preparations*—1 *Injectio Cocaine Hypodermica*, strength 10 per cent. *Dose*—2–5 m subcutaneously. 2 *Lamellae Cocaine*, each containing  $\frac{1}{10}$  gr 3 *Trochiscus Krameriae et Cocaine*, each containing  $\frac{1}{10}$  gr

The liquid extract of coca has been administered as a sedative and bitter in irritable stomach. Cocaine is used largely as a local anæsthetic, although within recent years it has been superseded for many purposes by other drugs having a similar local action and less general depressant effect. For anæsthesia by lumbar injection it has almost entirely been given up in favour of other substances, such as

stovaine and novocain The lozenges of krameria and cocaine are useful in sore throats. A 10 per cent solution is painted on the throat, if necessary, before making a laryngoscopic examination, and weaker solutions in the form of a spray are employed in painful laryngeal phthisis to enable the patient to swallow food In operations on the nose, throat, or any mucous surface a solution painted on gives complete local anæsthesia For operations on skin surfaces it must be injected with a syringe, and for this purpose a 2 per cent solution with the addition of a little adrenalin chloride is recommended A drop or two of a 5 per cent solution placed on the conjunctiva causes dilation of the pupil and is helpful in ophthalmoscopic examinations Solutions varying in strength from 2 to 10 per cent are employed for local anæsthesia in eye operations Cocaine has been given by the mouth in inflammation of the œsophagus, in cancer of the stomach, in gastralgia, and in sea-sickness The ointment is valuable in painful fissures, ulcers, etc, and in pruritis Suppositories and bougies containing cocaine are occasionally employed

## Cocaine.

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See ALKALOIDS (*Vegetable*), ANÆSTHETICS (*Local Anæsthesia*), ANALGESICS AND ANODINES, APPETITE (*Loss of*), COCA, COCAINA, GLAUCOMA (*Treatment*), HYPNOTISM (*in Cocaine Habit*), INSANITY, ETIOLOGY OF (*Exotoxic*), INSANITY, NATURE AND SYMPTOMS (*Etiological Varieties, Cocainism*), MORPHINOMANIA AND ALLIED DRUG HABITS (*Cocaine Habit*), NOSE, LOCAL ANÆSTHETICS, PHARMACOLOGY, PRESCRIBING SPINE, SURGICAL AFFECTIONS (*Spinal Cocainisation*), TOXICOLOGY (*Alkaloids and Vegetable Poisons, Cocaine*)

COCAINE is an alkaloid which was discovered by Niemann as long ago as 1860 in the leaves of *Erythroxylon Coca*, a shrub cultivated chiefly in Bolivia and Peru It was introduced into clinical use by Koller in 1884 In the British Pharmacopœia the official preparations are the dried leaves—Coca, extractum coca liquidum, cocaina, cocaine hydrochloras, lamellae cocaine, each of which contains  $\frac{1}{10}$  of a gram of the hydrochlorate of cocaine, injectio cocaine hypodermica, unguentum cocaine, and trochisci krameriae et cocaine

*The Alkaloid*—Cocaine is but slightly soluble in water. It dissolves readily in alcohol, in melted vaseline, in castor and other fixed oils, and in many volatile oils If heated in water it decomposes, and the solution contains ecgonine, benzoyl ecgonine, and benzoate of cocaine.

**The Salts.**—Hydrochlorate of cocaine, the official salt, is readily soluble in water, but insoluble in fats and oils.

**PHYSIOLOGICAL ACTION** — *Local* — When a solution of hydrochlorate of cocaine is applied to a mucous membrane it produces temporary local anaesthesia and anæmia, which last for ten minutes or more, according to the strength of the solution used. If applied to a turgescient mucous membrane, it produces collapse of the swollen structures, unless the turgescence has been so long continued as to result in connective-tissue hypertrophy. Temporary local dilatation of the blood-vessels may follow.

When applied to the conjunctiva, slight smarting, followed by anaesthesia, results. The pupil is dilated, an effect ascribed by Koller to contraction of the vessels of the iris, but by most other authorities to irritation of the sympathetic. Accommodation is impaired, but this passes off more rapidly than the paralysis produced by atropine.

The skin is much less affected by local application than are the mucous membranes.

**Internal.** — Internally cocaine acts as a stimulant, producing a sensation of exhilaration and well-being, with increased mental and muscular power. In animals the brain, medulla, and spinal cord are stimulated from above downwards. Large doses produce convulsions of cerebral origin. The sensory tracts of the cord are paralysed, and anaesthesia results. This effect has been produced in animals by the administration of dangerous doses, but quite recently Bier has produced a similar effect in the human subject by the injection of small doses ( $\frac{1}{2}$  to  $\frac{1}{4}$  gr) directly into the cerebro-spinal sac under conditions similar to those required by Quincke's exploratory puncture. He claims that complete anaesthesia can thus be safely produced below the level of the nipples. He has operated thus successfully on several patients chiefly for tuberculous bone and joint disease of the lower extremities.

**Circulatory** — Cocaine produces moderate stimulation of the heart's action.

**Respiratory** — Respiration is powerfully stimulated, and its rhythm is disturbed. Death from cocaine poisoning results from paralysis of respiration and the exhaustion produced by the accompanying convulsions.

Large doses produce a slight rise of temperature. Cocaine is mainly destroyed in the body, but may in part be eliminated by the kidney. The amount of urine is said to be slightly increased, and the urea to be diminished.

**METHODS OF APPLICATION** — The great value of cocaine lies in its property of producing local anaesthesia, and for this purpose various methods of application are adopted.

For the eye aqueous solutions of the hydrochlorate of the strength of 2 per cent, or even of 4 per cent, are made use of, a drop or two

of the weaker solution requiring to be applied two or three times at intervals of three or four minutes.

For the *mucous membranes* stronger solutions may be made use of even up to 50 per cent, but it is seldom necessary to use solutions stronger than 5 per cent. Several applications of the weaker solutions may be made at intervals of three or four minutes, or a small piece of cotton wool soaked in the solution may be left in contact with the part to be rendered anaesthetic.

For application to the *skin*, watery solutions are of little value, and for this purpose only solutions of the alkaloid should be made use of.

Injected hypodermically, anaesthesia is produced for a limited area around the puncture. A watery solution is made use of, and the dose as a rule should be limited to half a grain. Where possible, e.g. in amputation of a finger, a ligature should be tied round the part to be operated on in such a way as to obviate the risk of toxic effects from the passage of the drug into the system.

Another important method of application is that known as the infiltration method (Schleich). This is described in article "Anaesthetics," vol. i p. 159.

**Uses** — *Local* — The degree of anaesthesia produced by cocaine when applied to the eye is sufficient to allow of practically any operation being performed on the eyeball. It meets all the requirements that can reasonably be expected of a local anaesthetic, and hence has come to be regarded in ophthalmic practice as the anaesthetic *par excellence*.

In nasal, aural, and throat operations it is of great value, and the manner of application and other points of practical importance will be specially referred to under "Ear" and "Nose." In dentistry it is useful in toothache, and is also used to deaden the exposed pulp or dentine before filling the cavity of a carious tooth. By injecting a dose into the gum on each side of a tooth the pain of extraction is greatly lessened. By hypodermic injection or by Schleich's infiltration method nearly all minor surgical operations can be carried out painlessly.

During labour it has been used to relieve the pain of the dilating os uteri, and to relieve spasmodic contraction due to pain.

An oily solution of the alkaloid may be used to relieve the pain of shingles, eczema, neuralgia, pruritus, or urticaria.

Acute coryza, acute pharyngitis, hay fever, and similar conditions may be much relieved, especially in early stages, by a spray of a watery solution.

**Internal** — Cocaine is used internally for sea-sickness and the vomiting of pregnancy, as a tonic during convalescence, in mental exhaustion and muscular debility, and it has been recommended in alcoholism and the opium habit, but

its use in the latter cases must be strongly condemned on account of the risk of development of the cocaine habit

**DRAWBACKS TO THE USE OF COCAINE.**—One of the greatest dangers attending the use of cocaine is the development of the cocaine habit. This habit is frequently secondary to morphinism, but may be developed from the medicinal use of the drug. In one of several cases which have come under the writer's notice, the habit developed in a chemist who was wont to resort to a cocaine snuff to relieve a chronic nasal catarrh. The "coca wines" so recklessly pressed upon the public as useful "tonics" are also a source of danger (see "Morphinomania and Allied Drug Habits").

When used with caution untoward effects are not common, but such have occasionally occurred, especially after the too free application of solutions to the nose and throat. Symptoms noted have been giddiness, faintness, pallor, feebleness of the pulse, great dyspnoea, and final collapse into unconsciousness.

It is difficult to keep solutions sterile, and it must be remembered that solutions cannot be boiled without destroying the cocaine.

**EUCAIN.**—To obviate the disadvantages, and even dangers, occasionally attending the use of cocaine, efforts have been made to produce some substance free from these drawbacks. These have resulted in the introduction into practice of two synthetic products known as eucaine, both of extremely complex composition. For convenience these are distinguished as alpha-eucaine and beta-eucaine. The former of these was found to be less toxic than cocaine and to be an efficient anæsthetic, but, unfortunately, when applied to delicate mucous membranes it produced a great deal of smarting and irritation, and thus was unsuited for ophthalmological work. For beta-eucaine it is claimed that it is equal to cocaine in anæsthetic properties, that it is much less toxic, and that it does not cause local irritation. It is soluble in distilled water to the extent of 5 per cent, and the solution can be sterilised by boiling. Moreover, the solutions keep well.

Beta-eucaine may be used for all the surgical procedures for which cocaine is adapted, and in solutions of from 2 to 10 per cent. The stronger solutions must be made with hot water. For ophthalmological work a 2 per cent solution is recommended. It differs from cocaine in that it produces no dilatation of the pupil, and no disturbance of accommodation. Another point of difference is that it causes little or no contraction of turgid mucous membranes, a point which may be an advantage or a disadvantage according to what is desired.

**Cocainisation, Spinal.** See SPINE, SURGICAL AFFECTIONS OF (Spinal Cocainisation).

**Coccidiosis.**—A disease common in

young rabbits, due to the presence of the sporozoa, known as coccidia, in the liver and other organs, psoro-spermiasis. See PARASITES (Protozoa, Sporozoa).

**Coccidium.**—There are various species of coccidia, including the *C. hominis*, *C. cuniculi*, and *C. avium*; they are all parasitic and belong to the class sporozoa of the Protozoa; they infest rabbits and birds, and have occasionally been found in the liver and intestine of the human subject. See COCCIDIOSIS, PARASITES (Protozoa, Sporozoa).

**Cocculus Indicus.**—The dried fruits (Gr. κόκκος, a berry) of *Anemura paniculata*, which contain the poisonous glucoside (b) picrotoxin, which has the formula of  $C_{15}H_{14}O_8 \cdot H_2O$ , or  $C_{20}H_{24}O_{10}$  or  $C_9H_{10}O_4$ . See TOXICOLOGY (Alkaloids and Vegetable Poisons).

**Coccus.**—(1) Cochineal, or the dried fecundated female insect *Coccus cacti*, contains the glucoside carminic acid,  $C_{17}H_{18}O_{10}$  and carmine, and is used as a colouring agent (e.g. in the compound tincture of cardanoms). The official preparation is *Tinctura Cocci* (dose, 5 to 15 m). (2) A rounded or ellipsoid micro-organism (e.g. micrococcus, streptococcus, etc.).

**Coccyalgia or Coccygalgia.**—Pain in the coccygeal region. See COCCYGO-DYNIA.

**Coccygectomy.**—Excision of the coccyx. See COCCYGO-DYNIA.

**Coccygodynia.** See also COCCYGO-TOMY, LABOUR, INJURIES (Pelvic Articulations).—**Definition.**—A painful condition of the coccygeal region produced by the acts of sitting, walking, or defecation.

**Description.**—The structures involved may be the coccyx, sacra coccygeal joints and ligaments, perineal muscles attached to the bone, terminal branches of the sacral plexus of nerves, and probably the coccygeal gland. It has to be distinguished from a somewhat similar condition frequently associated with certain affections of the vulva, uterus, ovaries, pelvic peritoneum, and separation of the bones at the symphysis pubis. It is found almost exclusively in the female sex, although cases are recorded of its occurrence in males and children.

**Etiology.**—1 Traumatism during labour, causing—

(a) Injury to the fifth sacral and coccygeal nerves.

(b) Dislocation of the sacro-coccygeal joint, or fracture of the coccyx.

The latter may be produced by other causes, e.g. falls or blows.

2 Rheumatism affecting the ligaments or the periosteum of the coccyx.

### 3. Neuralgia affecting the terminal branches of the sacral plexus

Probably most cases are of this nature

**Diagnosis** should be made by placing one finger in the rectum and the other over the skin surface of the coccyx, when pain is produced on pressing the bone or structures round it, according to the part chiefly affected

**Symptoms**—Pain limited to the coccyx and neighbourhood, and aggravated by such acts as sitting or walking.

**Prognosis** is favourable, although many months may elapse before the pain completely subsides, and the condition is prone to recur

**Treatment**—Any concomitant pelvic disease must be first attended to. If there are indications of a primary inflammatory condition of the parts affected the patient should be kept at rest in bed, preferably in the lateral posture. Pain may be relieved by lead and opium fomentations, morphia hypodermically, or a suppository of morphia and belladonna.

Laxatives should be administered to facilitate defecation, which is frequently painful. In cases associated with dislocation or ankylosis of the sacro-coccygeal joints, massage and manipulation should be tried before resorting to operative measures. If there is no lesion of the bone the faradic current gives excellent results, one pole being applied over the sacrum and the other over the coccyx. As a rule two to twelve applications are sufficient. The application of the actual cautery over the origin of the sacral nerve may be resorted to if other means fail to give relief. The cases recorded as occurring in men and children have been successfully treated by anti-rheumatic remedies.

**Operative Treatment**—In obstinate cases Sir J. Y. Simpson recommended and carried out the subcutaneous division, by a tenotomy knife, of the muscular and fibrous tissues inserted into the sides and apex of the coccyx, with the object of preventing any movement of the bone. When the condition is associated with a lesion of the bone and unrelieved by other methods, Nott first suggested the complete extirpation of the bone. To carry this out a vertical incision is made over the posterior surface of the bone, the apex is then pulled well back, allowing the muscular attachments to be freed; finally, the bone is separated at the sacro-coccygeal point.

Both these latter methods are rarely necessary, and cases are recorded where the pain recurred even after operative treatment.

**Coccyx**.—The four terminal vertebrae or the single bone formed by their coalescence, the name is derived from Gr *κόκκυς*, a cuckoo, it being supposed that the bone has the shape of that bird's beak. See COCYGOODYNIA; LABOUR, PROLONGED; LABOUR, INJURIES TO THE GENERATIVE ORGANS (*Pelvic Articulations*),

RECTUM, DISEASES OF (*Kraske's Operation*), RECTUM, DISEASES OF THE (*Coccygeal Pain*)

**Cochin China Ulcer**. See SKIN DISEASES OF THE TROPICS (*Tropical Phagedena*).

**Cochineal**. See COCCUS.

**Cochlea**.—Part of the internal ear, a spiral cavity (Gr *κοχλίας*, a snail) in the osseous labyrinth (osseous cochlea), with membranous canals in it, one of which is named the scala media or membranous cochlea, and contains the organ of Corti. See BRAIN, PHYSIOLOGY OF (*Eighth Nerve*), PHYSIOLOGY, HEARING (*Internal Ear*).

**Cochleare**.—A spoon. See PRESCRIBING. This measure varies somewhat, containing from  $\frac{1}{2}$  fl dr to  $\frac{1}{2}$  fl oz of fluid, variations which are indicated by the expressions tea-spoon, dessert-spoon, and table-spoon. *Cochlearim* means "by spoonfuls."

**Cock's Operation**.—A method of performing external urethrotomy. See URETHRA, DISEASES OF (*Stricture, Treatment*).

**Cocles**.—Having one eye, monocolus, monophthalmus, derived from Gr *κύκλωψ*, a Cyclops or Round-Eye.

**Cocoa**.—The beverage made from the seeds of *Theobroma cacao*, also the fruit of *Cocos nucifera*. See DIET (*Beverages*), INVALID FEEDING (*Cookery in Diabetes, Cocoa-Nut Cakes*). PHYSIOLOGY, FOOD AND DIGESTION (*Tea, Coffee, Cocoa*).

**Cocoon Silk**. See DERMATITIS TRAUMATICA ET VENENATA (*Eczema, Causal Agents, Animal*).

**Codamine**.—One of the alkaloids ( $C_{20}H_{25}NO_4$ ) existing in opium (*q v*).

**Codeina**.—An alkaloid (methylmorphine,  $C_{17}H_{19}(CH_3)NO_3 \cdot H_2O$ ) obtained from opium or morphine. The dose of Codeina and of Codeinæ Phosphas is  $\frac{1}{2}$  to 2 grains. There is an official preparation of Codeinæ Phosphas, the *Syrupus Codeinæ*, which contains a  $\frac{1}{4}$  grain of the phosphate in each fluid drachm (dose,  $\frac{1}{2}$  to 2 fl dr). See ALKALOIDS, ANALGESICS, OPIUM, PHARMACOLOGY.

**Cod-liver Oil**.—*Oleum Morrhue* or the oil extracted from the liver of the cod (*Gadus morhua*). It has a complex composition, containing olein, palmitin, myristin, stearin, fatty acids (oleic, palmitic, and stearic), trimethylamine, traces of iodine and bromine, and alkaloids, such as morrhume ( $C_{19}H_{27}N_3$ ), aselline ( $C_{25}H_{32}N_2$ ), and gaduine. Its fishy smell prevents its external use, but it is given internally frequently in phthisis, tubercular affections, rickets, etc. The dose is 1 to 4 fl dr., and it

may be given as an emulsion, with malt, with iron, and in other ways See PHARMACOLOGY, PRESCRIBING, etc.

**Cælo- or Cello-.**—In compound words *cælo-* or *cælo-* (from Gr *κοῖλος*, hollow) signifies "relating to the abdomen." The *cæliac affection*, for instance, is an intestinal disorder of young children in which the stools are pale, bulky, loose, and porridgy, in which there is wasting, pallor, and absence of fever, and in which death is not an uncommon termination, although it may be long delayed. *Cœliadelphus* is a teratological type of double monster in which the twins are united by their abdomens. *Cœliagria* is gout in the abdomen. *Cœliochalasia* is a relaxed state of the abdominal walls. *Cœliocyesis* is ectopic pregnancy of the abdominal type. *Cœliodynna* is pain in the abdomen. *Cœliorchiis* is an open state (congenital) of the abdomen. *Cœliostegnosis* is constipation. *Cœliotomy* is opening into the abdominal cavity for diagnostic or operative purposes. See ABDOMEN, INJURIES OF (Treatment), LABOUR, OPERATIONS (Cæsarean Section), OVARIES, DISEASES OF (Ovariectomy), etc. *Cœlom* is the body-cavity or space between the two layers of the mesoblast. See FETUS AND OVUM, DEVELOPMENT.

**Cœnadelphus.**—From Gr *κοινός*, common, and *ἀδελφός*, brother—is that teratological type of united twins in which the heart or liver is common to both.

**Cœnæsthesia.**—The feeling of weariness and lassitude or of sprightliness, without the usual exciting causes (muscular work, etc.), or the vague consciousness of being, without the evidence supplied by the special senses.

**Cœnurus Cerebralis.**—From Gr *κοῖνός*, common, and *οὐρά*, a tail, the vesicular stage of the *Tænia cœnurus*. See PARASITES (Cestodes, *Tapeworms*).

**Coffee.** See CAFFEINIC ACID, CAFFEINE, DIET (Beverages), NEURASTHENIA (Treatment, Preventive), PHYSIOLOGY, FOOD AND DIGESTION (Tea, Coffee, Cocoa), TEMPERATURE (Diurnal Variations).

**Coffee-Ground Vomiting.**—The vomiting of blood altered in appearance by the action of the gastric juices. See STOMACH AND DUODENUM, DISEASES OF (Ulcer, Symptoms).

**Coffin-Birth.**—Birth of the infant after the death of the mother (*post-mortem parturition*), due probably to the presence of putrefactive gases in the abdomen.

**Coffinism.**—A mode of treatment of disease, so called after a Dr Coffin, who practised it, the drugs given were chiefly cayenne pepper and lobelia inflata.

**Cognac.** See ALCOHOL (Spirits, Brandy).

**Cogwheel Respiration.**—A jerky or wavy form of inspiration (in deep respiration) noted on auscultation in cases of pulmonary tuberculosis, etc. See CHEST, CLINICAL INVESTIGATION OF.

**Cohnheim's Theory.**—The theory that neoplasms originate in cell "rests" present in the tissues before birth and lying latent till adult life, it was supposed that the discovery of the microbic origin of some tumours had discredited this theory, but in its modified modern form (theory of embryomata) it has many supporters. See also ADRENAL GLANDS (Tumours, Adrenal "Rests").

**Colf.** See CAUL.

**Colling of Cord.** See LABOUR, FAULTS IN THE PASSENGER (Cord), LABOUR, ACCIDENTAL COMPLICATIONS (Coils of the Cord).

**Coin-Sound.**—The metallic echoing sound heard in pneumothorax, when one observer listens over the back of the chest while another sharply taps a coin placed on the front of the chest. See BRUIT D'AIRAIN.

**Coitus.**—Sexual intercourse (from Latin *cœo*, to come together). See MEDICINE, FORENSIC (Rape), PREGNANCY, DIAGNOSIS (Date of Coitus), PREGNANCY, MANAGEMENT (Coitus during Pregnancy), VICE (Sexual System, *Apathy*).

**Coke.** See TOXICOLOGY (Gaseous Poisons, Carbon Monoxide).

**Cola.** See KOLA.

**Colchicine.**—An alkaloid (?), the active principle of colchicum (*q v*), ebullition with acidulated water is said to convert it into colchicine ( $C_{21}H_{22}(OH)NO_6$ ) and methyl alcohol, according to *Reisel* the formula of colchicine is  $C_{21}H_{22}(OCH_3)NO_6$ . See ALKALOIDS (Vegetable), COLCHICUM, TOXICOLOGY (Alkaloids, Colchicum).

**Colchicum.** See GOUT, PHARMACOLOGY, PRESCRIBING, TOXICOLOGY (Colchicum).—Both the corn and the seeds of *Colchicum autumnale* are official. The active principle is *Colchicine*, a yellow crystalline alkaloid. *Veratrine* is also present in traces. The seeds contain a greater proportion of the active alkaloid than the corn, and possess in addition a volatile oil. The preparations from *Colchici cornus* are—1. *Extractum Colchici*. Dose— $\frac{1}{4}$  gr. 2. *Vinum Colchici*. Dose—10-30 in. From *Colchici semina* is prepared *Tinctura Colchici Seminum*. Dose—5-15 m.

Colchicum is a specific for gout, and is hardly ever used except in this disease. Given during an acute attack, it lessens the pain and cuts short the attack. In smaller doses in the

intervals it lessens the severity and diminishes the frequency of the seizures. It is also used in a great variety of conditions which are, or are supposed to be, of a gouty nature. It has been recommended in small doses as an addition to an aperient pill in chronic articular rheumatism. The beneficial effects of this drug probably depend on an increased excretion of toxic products resulting from stimulation of the hepatic and intestinal functions.

**Cold.** See ANTIPYRETIC and ANTIPYRETIC MEASURES (*Application of Cold*), HEMORRHAGE (*Local Treatment, Cold and Hot Water*), HYDROPATHY (*Cold Pack*), MEDICINE, FORENSIC (*Death from Cold*), PUERPERIUM, PATHOLOGY (*Parametritis and Peritonitis, Ice-bag*), STOMACH AND DUODENUM, DISEASES OF (*General Etiology, Cold and Dump*), TEMPERATURE (*Treatment of Fever, Cold Bath*)

**Cold Cream.**—Cold cream is the Unguentum Aquæ Rosæ, an official preparation of the Oil of Rose (q.v.)

**Cold Pack.** See HYDROPATHY (*Paralysis, Cold*)

**Cold Spots.** See PHYSIOLOGY, SENSES (*Temperature Sense*)

**Colectomy.**—Excision of a part of the colon. See COLON

**Coleoptosis.**—Prolapse of the vaginal walls with or without prolapse of the uterus (Gr *κολεός*, vagina, and *πτῶσις*, descent)

**Coleorrhexis.**—Rupture of the vagina (Gr *κολεός*, vagina, and *ῥήξις*, rupture)

**Coleostegnosis.**—Narrowing or constriction of the vagina (Gr *κολεός*, vagina, and *στέγνωσις*, constriction)

**Coley's Fluid.**—A mixture of the streptococcus of erysipelas with bacillus prodigiosus, grown together in the same broth, and injected in cases of malignant growth (e.g. sarcoma) when operation is impossible. See THERAPEUTICS, SERUM THERAPY (*Coley's Fluid*), TUMOURS, INOPERABLE, TREATMENT OF (*Bacterio-Therapy*)

**Colic.** See APPENDIX VERMIFORMIS (*Appendicitis, Symptoms*), DEVONSHIRE COLIC, GALL-BLADDER AND BILE DUCTS, DISEASES OF (*Gall-Stones*), GASTRO-INTESTINAL DISORDERS OF INFANCY (*Digestion, Flatulence, and Colic*), KIDNEY, SURGICAL AFFECTIONS OF (*Movable and Floating Kidney, Pain*), LIVER, DISEASES OF (*Hepatoptosis, Symptoms*), MYIASIS (*Myiases Intestinalis*), ŒSOPHAGUS (*Inflammation*), PANCREAS, DISEASES OF (*Cyst, Symptoms*), TOXICOLOGY (*Chronic Lead Poisoning*), TRADES, DANGEROUS (*Lead Poisoning*)—Under normal conditions the muscular

coat of the digestive tract performs its functions of mixing and propelling food from the stomach downwards, and of finally expelling the fæces, painlessly, but, under numerous abnormal conditions, its gentle, painless, and harmonious working may be roused into violent, painful, and irregular action, the symptoms of which are designated by the term colic, from the Greek *κόλον*.) Colic, as ordinarily understood, may therefore be defined as abdominal pain due to spasmodic and painful contraction of the alimentary musculature. Other hollow tubes or hollow organs may be the seat of similar morbid action, and may exhibit like symptoms, and the original term has been extended to the gall-bladder and bile-ducts (hepatic colic), and to the kidney and ureter (renal colic). But this article will deal solely with colic as occurring in the musculature of the digestive tract.

**SYMPTOMS.**—The essential and outstanding symptom is pain, located in the abdomen, remittent or intermittent, withing or twisting in character, and generally relieved by pressure. Its onset may be sudden or preceded by premonitory uneasiness. It may be localised to a particular part of the abdomen, but if severe, its origin cannot be defined, and it is felt generally over the abdomen with its maximum around the umbilicus. It is spasmodic, coming in waves, shorter or longer in duration, and rising from the level of complete absence of suffering, or of more or less persistent uneasiness. It varies in severity from bearable griping to agonising spasm, and in duration from a few minutes to several days. As in all abdominal pain, the patient intuitively flexes the trunk and draws up the limbs, but, as pressure generally relieves rather than aggravates his suffering, he indulges the desire to move or roll about, at the same time applying pressure in various ways, such as rubbing by hand or leaning against a pillow. Where there is considerable bowel distension or associated inflammation, pressure aggravates the suffering. The pain is of a peculiarly depressing character, the face is pale and indicative of suffering, the skin is cool and possibly moist, the pulse is normal, or weakened and slowed rather than quickened, the temperature is not raised, the bowels may be seen, felt, and heard to be in excited peristalsis, vomiting may or may not be present, the bowels may be obstinately confined, or enabled sooner or later to expel their gaseous or other contents with gratifying relief to the distressing symptoms. If the cause of the colic be towards the anus the pain is associated with a bearing down and expulsive desire. Vomited matters are at first from the stomach, but eventually, when the cause of the colic is bowel obstruction, they are from the bowels. The abdomen may be retracted or distended according to the amount and character of the bowel contents, and the condition of the intestinal and abdominal

**musculature** The breathing is interfered with according to the degree of associated contraction of the respiratory muscles

**Pathology**—The essence of colic is excessive contraction of the non-striated fibres that form the alimentary musculature, and it has, as its analogue, the tetanic contraction of striated muscle that gives rise to cramp. The alimentary canal possesses within itself, independently of the central nervous system, the nervous mechanism necessary for peristalsis which can be directly stimulated. But the bowel movements are also influenced and secured by stimuli acting through the motor or augmentor vagus, reflexly or from the cerebral centres. Hence the abnormal or excessive stimuli that lead to colic may be local, as they generally are, or they may be distant, acting reflexly or directly from the cerebral centres. The pain is not a neuralgia in the ordinary sense of the term, but is caused by pressure on nerves from severe muscular contraction. As the splanchnic nerves inhibit peristalsis they can have no causative influence on colic.

The inclusion of visceral neuralgia under the term colic is undesirable.

**Diagnosis**—The diagnosis of colic would be comparatively easy were it not frequently associated with ailments which complicate and obscure its manifestations. It must be diagnosed from other diseases, from other colics, and colic occurring in one part of the alimentary canal must be differentiated from colic in another.

1 From other diseases—

(a) *Inflammation*—In inflammation the chief distinguishing symptoms are that the pain is increased on pressure, that it is more or less constant, and that fever is present as shown by quickened pulse, hot skin, and raised temperature.

(b) *Neuralgia*—In neuralgia the pain is shooting or stabbing in character, tends to dart in various directions, to be increased rather than relieved by pressure, and to be associated with superficial hyperæsthetic areas. There is a history sufficient to account for lowered health, and there are present other signs of the neurotic temperament.

(c) *Angina Pectoris*—Certain cases of angina pectoris are difficult to distinguish from abdominal colic, and have to be kept in view in making a diagnosis.

2 From other colics (Hepatic, Renal, Bladder, Uterine) —

(d) In *hepatic colic* the pain is in the upper part of the abdomen, and is towards the right side and right shoulder. Excited gastric or intestinal peristalsis is absent. There may be the history of previous attacks. After an attack there is tenderness on pressure over the gall-bladder. There may be jaundice, and, if gall-stones be expelled, they are to be found in the stools.

In *renal colic* the pain is in one or other flank ;

it shoots down towards the bladder and into the penis and testicle, it is associated with frequent desire to micturate, and possibly with the expulsion of gravel or calculi.

In *strangury* the pain is confined to the lower part of the abdomen, and is associated with urinary urgency.

In colic in the unimpregnated uterus the pain is in the hypogastrium, and it occurs in relation to the *menstrual period*.

The physiological colic of an impregnated uterus needs only to be referred to.

In *Fallopian tube colic* (one manifestation of Mittelschmerz) pain occurs in one or other ovarian region, in mid-menstrual period, there may be sudden, clear, leucorrhœal discharge, and fullness at painful side may be found on vaginal examination.

3 Colic in the alimentary canal may be more or less confined to a particular portion of its area.

In *gastric colic* pain and distension occur in the upper central part of the abdomen. The percussion note is stated to be more prolonged and of a lower pitch than over the colon. The characteristic intestinal movements are absent. Eructation of gas attended by relief, also vomiting, more freely occur.

In *intestinal colic* the symptoms previously described are characteristic, colic in the lower bowel being specially attended by tenesmus.

*Appendicular colic*—In a typical case it occurs at somewhat regular intervals, say, from three weeks to three months. While at its height the pain is felt over the abdomen generally, it begins and ends in the right flank, tenderness to pressure being located in the region of the appendix. Vomiting occurs, and the patient may imagine he suffers from bilious attacks, all the more that they may recur for a period of years even. The attack lasts for a few hours, and is not in the first instance associated with feverishness. But sooner or later inflammation complicates the situation. The writer believes from clinical experience that appendicular colic is comparatively frequent, but that as many of the attacks are mild, they are unrecognised by patient and physician alike. Being mild, the operating surgeon sees nothing of them, in severer cases he deals with them only after inflammation has complicated and obscured the clinical picture.

*Etiology*—Colic, like cough, being a symptom rather than a disease, is caused by and is associated with numerous morbid conditions. It is the expression of a normal function acting in excess in response to undue stimulation, and having as its main object the overcoming of unusual difficulty or the expulsion of an irritant.

In certain individuals the intestinal musculature is more easily stimulated to excessive action than in others. There is a predisposition to

colic, the same degree of stimulus having vastly different effects in different individuals. The exciting causes may be within the alimentary tube, in its wall, or outside altogether. The contents may act by their quantity or by their quality, or by both. Mere quantity by over-distension will over-stimulate, e.g. accumulation of gas or of faeces from constipation, or from the various forms of obstruction, such as simple and malignant stricture, intussusception, volvulus, internal strangulation from bands, peritoneal adhesions and kinks, intestinal concretions, large gall-stones, coiled-up bundles of worms, and hernia. Exciting quality of contents is exemplified by irritating articles of diet, themselves indigestible or containing ptomaine poison, by irritant poisons, by irritating purgative medicine, by irritating results of evil digestion. Exciting causes situated in the intestinal wall are seen in local injury, as from intussusception, hernia, etc., acting primarily by direct local effect, and, secondly, by over-distension behind, in ulceration, malignant, simple, tuberculous, or dysenteric, in inflammation, catarrhal, enteric, or peritonitic, where the pain is partly to be accounted for by excited peristalsis. Causes outside the intestinal canal find their illustration in exposure to cold, in dentition, in mental anxiety or emotion, in disease of the spinal cord as locomotor ataxia, but these are associated rather with diarrhoea or neuralgia than with colic.

It has not yet been satisfactorily demonstrated how the *poison of lead* produces colic. It probably does not act through constipation or through organic disease of the sympathetic, but whether it acts directly on the intestinal musculature or nervous ganglia, or by leading to contraction of the blood-vessels, is a moot question.

**TREATMENT.**—Whatever the cause, pain demands relief, all the more that, if very severe, it may lead to serious collapse, while the excited peristalsis, of which it is a symptom, has been found to cause rupture of the bowel. When intense, morphia should be injected subcutaneously, and winffs of chloroform given until it has had time to act. When less severe a hot bath or a warm poultice applied over the abdomen, with some sedative and antispasmodic, as morphia and chloric ether internally, suffices. Pain being relieved there is time to examine quietly into the case and to determine the cause, and upon the cause depends the scientific and successful treatment of the condition. The sedative, moreover, when given in an appropriate dose, paves the way by quieting irregular spasm for that regular and harmonious action of the musculature which leads to success. In gastric colic the stomach must be emptied. If it fail to do so by its own efforts, emesis must be encouraged, or the stomach tube used.

In intestinal colic of any gravity, obscurity, or permanence, the physician should, without

undue delay, associate himself with a surgeon. Where mere mechanical causes can be excluded, such as hernia, volvulus, etc., nature's attempt to empty the bowels must be assisted by the immediate use of rectal injections, and by the administration, where vomiting is not actively present, of non-irritating purgatives, such as castor oil, calomel, or salines.

Where faecal accumulation is within reach it is generally necessary to combine the use of the finger with the action of the enema in order to get rid of it. When legitimate efforts have failed to empty the bowel and give permanent relief, there should be no undue delay in deciding the question in consultation with a surgeon, whether the abdomen is to be opened or not for the detection and removal of any obstruction, or for the making of an artificial anus above the recognised seat of obstruction.

Appendicular colic must be dealt with by removal of the appendix in a quiet interval.

Until relief is obtained, in acute cases it is practically useless to feed the patient. Ice to suck or hot water to sip in order to relieve thirst should be given.

Where recurring attacks occur from mal-digestion, as in bottle-feeding of infants, prevention must be secured by proper dieting.

Where colicky attacks are caused by catarrh or by ulceration of the bowel, appropriate dietetic and medicinal treatment is necessary.

The colic said to be associated with the first stage of peritonitis, and the colics from causes outside the alimentary canal, acting reflexly, can only be relieved by sedative treatment such as morphia or the hot bath.

**Colica Pictorum.**—Lead colic, especially common in Poitou (hence the name).

**Colitis.**—Inflammation of the mucous membrane and also of the deeper-seated parts of the colon. See COLON, DISEASES OF. See also APPENDIX VERMIFORMIS (*Appendicitis, Diagnosis*), CHOLERA NOSIVRA (*Diagnosis*), FACIES (in *Ulcerative Colitis*), SROOIS (*Intestinal Sand and Gravel*), TYPHOID FEVER (*Diagnosis from Ulcerative Colitis*).

**Collagen.**—A substance of which non-elastic fibres are composed, allied to the proteids (but not yielding tyrosin when decomposed), having a great affinity for carmine, when boiled it takes up water to form gelatin (Gr *κόλλα*, glue, *γαινάρ*, to form). See PHYSIOLOGY, TISSUES (*Connective Tissues*).

**Collapse.**—The loss, more or less sudden, or the great weakening, of most of the signs of vital activity, occurring either as the last stage of shock or as the result of a severe disease (e.g. cholera) or a long-continued one (typhoid fever). See SHOCK (*Definition*). See also CHOLERA, EPIDEMIC (*Treatment, Collapse*), LABOUR, POST-



**PARTUM HÆMORRHAGE** (*Post-hæmorrhagic Collapse*), LABOUR, INJURIES TO THE GENERATIVE ORGANS (*Rupture of Uterus*), LABOUR, INJURIES TO THE GENERATIVE ORGANS (*Acute Inversion of Uterus*), PANCREAS, DISEASES OF (*Hæmorrhage in*), PUERPERIUM, PATHOLOGY (*Sudden Death*), TEMPERATURE (*Alterations, Depression*)

**Colles' Fracture.** See WRIST-JOINT INJURIES (*Fractures of the Bones of the Fore-arm*)

**Colles' Law.**—The fact that a woman (not apparently suffering from syphilis) who has given birth to a syphilitic child may suckle it without risk of becoming infected, it is supposed to be due to maternal immunisation by small doses of the syphilitic virus received through the placental connections, it is not an absolute law, for exceptions have been reported (*G. Novy, Zentralbl. f. Gynak.* xxx 590, 1906) See BRAUNER'S LAW, PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Syphilis*)

**Colley's Operation.** See PALATE, CLEFT (*Operative Treatment*)

**Collier's Bronchitis.** See BRONCHI, BRONCHITIS (*Chronic*)

**Colliquative Necrosis.**—A post-necrotic tissue-change in which softening and liquefaction without decomposition occur, *e.g.* in the cerebral tissues after embolism

**Collodia.**—Solutions of pyroxylin or soluble gun cotton ( $C_6H_5(NO_2)_3O_2$ ) in ether or in a mixture of ether and alcohol. See COLLODION

**Collodion.**—The official *collodium* is a solution of dimnitro-cellulose ( $C_6H_5(NO_2)_2O_2$ ) in pyroxylin, in ether and alcohol, from this, *collodium flexile* is obtained by the addition of Canada balsam and castor oil. *Collodium vesicans* contains 1 part of pyroxylin dissolved in 40 parts of Liquor Epispasticus. Collodion, when painted on the skin, rapidly dries and forms a protective film, so it is used for closing small wounds, for attaching dressings, for protecting irritated parts from the air, and for the local application of medicines, such as cantharides, carbolic acid, iodoform, mercury, creosote, iron, lead, tannic acid, etc. See ASEPTIC TREATMENT, COLLOID, GOSYPIUM, PHARMACOLOGY, PRESCRIBING

**Colloid Degeneration.**—The formation of a semi-solid, structureless, jelly like substance (colloid) in epithelial cells, especially in those of the thyroid gland and in cancers, colloid has no fixed chemical constitution, but it is not identical with mucin (for it is precipitated by tannic acid, but not by alcohol and acetic acid). See CHOROID, DISEASES OF (*Choroidal Degeneration*), FLUIDS, EXAMINATION OF (*Ovarian*

*Cysts*), PERITONEUM, TUMOURS (*Colloid Cancer*), STOMACH AND DUODENUM, DISEASES (*New Growths, Carcinomata*).

**Colloid Millum.** See TUMOURS OF THE SKIN (*Benign, Colloid-Millum*)

**Colloid, Styptic.**—A preparation containing tannic acid (20 parts), alcohol (5 parts), stronger ether (20 parts), and collodion (55 parts), xyl-styptic ether (U S Pharmacopœia).

**Collunaria.**—Nasal douches or washes (Latin, *colluo*, I wash)

**Collutorium.**—A mouth wash or gargle (Latin, *colluo*, I wash).

**Collyria.**—Eye-salves or eye-washes (Gr. *κολλῆριον*, an eye-salve)

**Coloboma.**—Coloboma (Gr. *κολοβός*, mutilated) signifies a defect, and more especially a congenital fissure or defect of some part of the eye, *e.g.* of the eyelid (*C. palpebræ* or *blepharocoloboma*), of the iris (*C. iridis*), of the choroid (*C. choroidis*), of the lens (*C. lentis*), or of the retina (*C. retinæ*). Fissure of the lobule of the ear is known as *Coloboma lobuli*. See CHIRIA, FISSURE OF, CHOROID, DISEASES OF (*Congenital Affections*), EYELIDS, AFFECTIONS OF (*Congenital Defects*), IRIS AND CILIARY BODIES (*Congenital Abnormalities of the Iris*), LENS, CRYSTALLINE (*Coloboma*), MENTAL DEFICIENCY (*Coloboma iridis in*), PALATE (*Congenital Malformations of Mouth*), RETINA AND OPTIC NERVE (*Congenital Abnormalities*)

**Colocynth.** See PHARMACOLOGY, TOXICOLOGY (*Abortifacients*)—*Colocynthis Pulpa* is derived from *Citrullus colocynthis*, the bitter apple. It contains *colocynthin*, a neutral crystalline glucoside, and various resinous substances. Its preparations are—1 Extractum Colocynthis Compositum. Contains also Barbados aloes and scammony. Dose—2-8 gr. 2 Pilula Colocynthis Composita. Contains also Barbados aloes and scammony. Dose—4-8 gr. 3 Pilula Colocynthis et Hyoscyami. Same formula as the last with extract of hyoscyamus added. Dose—4-8 gr.

Colocynth is a very efficient and widely used hydragogue cathartic. It causes a large increase of intestinal secretion, and stimulates the whole length of the bowel. It causes considerable griping if given alone, and the pill with hyoscyamus is therefore most commonly employed. It is too irritating to be given repeatedly over long periods, but whenever a free purging of the whole intestinal tract is required no better drug is to be found.

### Colon, Diseases of.

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See also ABDOMEN, INJURIES OF (*Lesions of the Intestine*), GASTRO-INTESTINAL DISORDERS OF INFANCY (*Congenital Dilatation of the Colon*), INTESITINES, SURGICAL AFFECTIONS OF (*Strictures*), LIVER, TROPICAL ABSCESS (*Rupture into Colon*), PHYSIOLOGY, FOOD AND DIGESTION (*Large Intestine*); TYPHOID FEVER (*Symptoms*, "*Bell Sound*")

**INFLAMMATION AND ULCERATION — 1. SIMPLE COLITIS.**—The colon may be the seat of a simple catarrhal inflammation comparable to a gastritis or a bronchitis. If in such a case the inflammation extends low enough, a rectal examination with a speculum will show the mucous membrane to be intensely injected, of a light red colour, swollen, and secreting a thick mucus. The description of Wilks and Moxon gives a very good picture of the state of things seen after death. They mention a "case attended by discharge of mucus and blood where after death the whole internal surface of the colon presented a highly vascular soft red surface covered with tenacious mucus or adherent lymph, and here and there showing a few minute points of ulceration. The coats also were much swollen by exudation into the mucous and sub-mucous tissues."

There are two varieties of simple colitis, viz that which occurs either by extension from neighbouring parts or in association with some grave morbid condition, and that which is unassociated with any other serious malady. This, which has been called *acute primary colitis*, will now be described.

**Symptoms.**—The main symptom is diarrhoea, which may come on suddenly, there is much mucus in the stools, and often blood also, even in considerable quantities. At first there may be absolutely no faecal matter, but as the patient improves the motions contain more feces and less mucus. The blood is mostly fluid and but little changed, so that we may infer that it has but recently left the vessels, and has come, therefore, from the large intestine. Often the mucus is in little lumps. Triple phosphate crystals may be seen on microscopical examination, and, more rarely, oxalate of lime, cholesterolin, and Charcot's crystals. The bowels may be open many times a day, and although tenesmus is not a striking feature, it may be present. A rectal examination reveals nothing abnormal except that the mucous membrane may feel a little rough. Abdominal pain is a very common symptom, it comes on in paroxysms, often

associated with defæcation, but it bears no relationship to food. It is of a gripping character, often very severe, and it nearly always follows the course of the colon. Between the attacks the patient may be free, but he sometimes complains of a dull pain. Abdominal tenderness is usually present, a very common seat for it is over the *sigmoid flexure*, but the whole colon, or even the whole abdomen, may be tender. There may be considerable pyrexia even when there is much diarrhoea and loss of blood. The pulse is rapid, and in a severe case small, soft, and running. The abdomen is not much distended, and sometimes nausea, vomiting, and loss of appetite are present. The tongue is furred, and the fur is nearly always a pure white, while in many diseases with which colitis might be confounded it is a brownish white. There is often much mental depression, the sufferer from this disease exaggerates trifles and takes a gloomy view of life. In some cases the neurotic element is very marked, and we are forcibly reminded of the chronic neurotic dyspeptic. Many of these neurotic patients are men who may be said almost to live for their illness, and they are usually most difficult to cure.

Simple colitis is by no means always so severe as this description might lead the reader to expect, but the difference is only one of degree. The majority of cases are mild, but, on the other hand, the diarrhoea may be uncontrollable, and the patient may die from exhaustion.

The *treatment* consists in keeping the patient absolutely in bed until the diarrhoea has stopped, the motions are well formed, blood is no longer passed, and the temperature is normal, and longer still if he has had a severe attack. Warmth to the abdomen is very desirable. He should consume nothing but milk, and should not take more than two fluid ounces at a time. The total daily amount will depend upon the acuteness of the attack and the general condition of the patient. A good way of checking the diarrhoea is to give some laudanum or chlorodyne with fifteen or twenty grains of carbonate of bismuth suspended in some mucilage every four hours, the compound kino powder is also very valuable. If these drugs fail, a starch and opium enema will often succeed. Should opium be for any reason contra-indicated, compound catechu powder is very useful. But all astringent drugs should be omitted as soon as possible, for the constipation that follows colitis is often very troublesome. It is best relieved by a rectal injection of six or eight ounces of warm olive oil or a drachm of glycerine. A long holiday among new and interesting surroundings greatly aids the convalescence in neurotic cases.

The *diagnosis* is not usually difficult. The disease is known from *ulcerative colitis* by its sudden onset, by the large amount of mucus in the motions, by the fact that blood appears

early, and also by the fact that under appropriate treatment the case usually yields. In England *acute dysentery* is hardly likely to lead to a mistake, but the meat-washing character of the stools, the burning pain in the rectum, the intense tenesmus, and the constant desire to go to stool even when nothing is passed, should prevent mistake. It is, however, important to remember that in England we often see severe cases of colitis in persons who have had dysentery abroad, and under strict treatment these cases recover completely. *Enterica, malignant disease of the bowel, and arsenical poisoning* may all give rise to errors of diagnosis.

The *prognosis* is as a rule good, and acute cases are soon well, but in cases that have been allowed to become chronic—and often the disease has lasted many months before it is taken seriously in hand—the patients require rest in bed and milk diet for many weeks before recovery is complete, and it should be remembered that few patients are kept in bed too long, but that many are not kept in bed long enough. The more neuritic a patient is, the worse is the prognosis, and some neuritic women seem never to lose the disease in spite of the most careful treatment.

That colitis which occurs either by extension from neighbouring parts or in association with some grave morbid condition need not detain us long, for it is generally marked by the diseases of which it forms a part. I have come across the following varieties—

I have seen the colon actually inflamed in a case in which there was *acute gastritis* due to the swelling of the acid. The stomach was in contact with the colon, and the colitis appeared to be due to direct extension of inflammation, for all coats were implicated.

Sometimes direct irritation, such as *enormous doses of purgatives*, will cause colitis, and in some cases of *arsenical or mercury poisoning* colitis is found which is probably due to excretion of the metal into the large intestine after it has been absorbed higher up in the alimentary canal.

Acute colitis may be associated with *septic or pyæmic conditions*. As an instance I may mention the case of a woman who had pelvic abscesses and peritonitis following gonorrhœa. She died from exhaustion. In the cæcum and for two feet beyond it the mucous membrane was sloughing, and from beyond this to the anus the colon was acutely inflamed, its walls were oedematous and thickened, its mucous surface was greyish yellow with patches of submucous hemorrhage; the mucous membrane was separating in shreds in many places.

Acute colitis is a rare complication of *Bright's disease*, and it may be that this form should be regarded as the early stage of ulcerative colitis, which is a recognised complication of Bright's disease. This form of acute colitis is often over-

looked because the diarrhœa due to it may be attributed to the purgatives so often given in Bright's disease, to uræmia, and very little stress can be laid upon blood in the motions, because patients with Bright's disease may bleed from their intestinal tract.

Colitis may in very rare cases be associated with *pneumonia*, and occasionally a *lardaceous colon* becomes acutely inflamed.

**2 MEMBRANOUS COLITIS**—Two distinct varieties exist, one which might be termed *dyspeptic membranous colitis*, and another which is always associated with some other grave condition, or is due to direct injury, and therefore might be called *secondary membranous colitis*. It is not usual to include under membranous colitis those cases of constipation in which, when the bowels are open, shreds of coherent mucus are passed with or without hard masses of fecal matter, such cases are common enough.

Dyspeptic membranous colitis is so called because the sufferers from it complain much of dyspepsia and they pass membranes from the anus.

The patients are usually over twenty years of age, the disease is commoner in women than in men, and in private than in hospital practice. In children it is excessively rare, for Edwards found that out of 111 cases only 6 were under the age of ten. The distinguishing feature of it is that membranes, which the patient usually calls skins, are passed from the anus. They are usually white, but may be brown from fecal staining. In extreme cases the membrane forms a complete tubular cast of the intestine, usually varying from one to six inches long, but such tubes have been known to be much longer, and sufficiently thin and tenacious to permit of their being held up. The wall of the tube may be laminated, and feces may be found between the laminae, showing that they have been laid down at intervals. Fecal matter may also be found in the tubes, the diameter of which may be anything up to 1½ inch. The thickness of the wall may be anything up to a quarter of an inch. Either with or without the tubes, shreds of all shapes and sizes may be passed, but in essential characters they are the same as the tubes. Often several pieces of membrane are passed rolled up into a ball. Under the microscope the membranes are structureless and transparent, embedded in them may be seen minute fragments of food and feces, some cells, free nuclei, microorganisms, phosphates, and cholesterol crystals. The cells are apparently the epithelial cells of the intestine that have undergone fatty degeneration. When the inner surface of the membrane is magnified it appears reticulate, and presents at regular intervals depressions or even perforations that clearly correspond to Lieberkuhn's follicles, and sometimes it is evident that the cells lining the crypts have been cast off with them. Chemically the membrane consists

of mucus, and is a coagulated secretion of the intestinal mucous membrane

The patients who suffer from this disease are usually neurotic dyspeptics of a depressed turn of mind, and liable to attacks of constipation. They are poor eaters, believing that first this and then that article of food disagrees with them, so that their diet soon becomes very restricted in choice and quantity, they are usually thin, anemic, and complain of the cold, the tongue is pale and a little furred, the bowels are frequently rather constipated, and the constipation may alternate with attacks of diarrhoea. Sufferers from membranous colitis are as a rule taciturn, they rarely have buoyant spirits, they take a gloomy view of life, and exaggerate the importance of trifles. These symptoms and this frame of mind are constantly with the patient, but there are exacerbations from time to time during which there is much additional pain, usually griping, generally along the course of the colon, and often coming on a long while after food. During these exacerbations flatulence is troublesome, loss of appetite and constipation are very marked, the patient may complain of nausea or suffer from actual vomiting, the tongue is very furred and may be red, and the mental depression and feeling of weariness are very pronounced. After this state of things has gone on for a week, the patient notices that she has passed a considerable quantity of "skins." In a mild case the patient is in the intervals between these attacks restored to feeble health without the passage of any membrane, but in other cases some but less membrane is passed in between these exacerbations of the disease. When passing membranes the patient is as a rule excessively constipated, and frequently takes enormous quantities of purgatives. The act of defecation is often excessively painful. I have known a patient sit on the water-closet for a couple of hours suffering from agonising griping pain before a motion, consisting of enough blood and membranes to fill a half-pint measure, was passed. This patient often had to take a morphia injection during defecation, and twice she fainted in the water-closet. As a rule a rectal examination reveals nothing abnormal. Only in mild cases does the opening of the bowels relieve the symptoms from which the patient suffers.

Women who complain of membranous colitis are particularly prone to suffer from amenorrhoea and pelvic troubles, indeed, almost every case that consults a general physician has been previously under the care of a gynecologist. Most of these patients, if severely ill, are valetudinarians who drag out their lives passing from one health resort to another, and many of them have enteroptosis or prolapse of the intestines.

*Treatment.*—Between the attacks the patient should in all respects lead as healthy a life as possible. She should take plenty of exercise.

Riding or playing golf is infinitely preferable to dull solitary walks taken merely for the sake of taking exercise. The diet should be ample, most of these patients decline first one article of food and then another, until at last they are not only underfed, but their digestive powers are overtaxed in one particular direction. An obviously indigestible dietary should be avoided, but otherwise the patient should partake of whatever is put before her. It should be well cooked, and the meals must be as tempting as possible, they should be at regular times. Van Noorden thinks that there should be plenty of coarse vegetables and fruit so as to mechanically act upon the colon. The patient should go to bed early, and have eight hours' sleep. All these patients are worse if they are idle, they should always have some occupation. Purgatives should be avoided, for they are particularly liable to cause indigestion, the bowels should be kept open by going to the water-closet regularly at the same time every day, by healthy living, and if necessary by a little abdominal massage before rising in the morning. A holiday and change of scene to some such bracing place as Switzerland or Norway is often of the greatest benefit. Indeed, these patients derive much benefit from such a holiday even when they are not passing membranes. During the period when they are passed it is particularly important not to give purgatives by the mouth, but if the constipation be extreme and the griping pain severe, considerable relief may be afforded by a rectal injection of a drachm of glycerine or half a pint of warm oil or soap and water. For a severe case rest in bed, morphia injected subcutaneously, and the application of hot fomentations to the abdomen, may be necessary, but opiates should not be prescribed unless they are absolutely necessary, for not only do they increase the constipation, but sufferers from membranous colitis are just the sort of people who may have become addicted to an excessive use of these agents.

Probably future experience will show that when the patient has tried all other means of relief without success, and when suffering is so great that life is a burden, that the best treatment is to open the colon on the right side, and thus give the diseased bowel complete rest. The artificial anus may then be closed. I believe the first published case in which this treatment was adopted was one brought before the Clinical Society by Mr. Golding-Bird and myself in 1895, and since then other authors have published cases and Mr. Golding-Bird and I have brought two others before the Clinical Society. The small experience we have at present of this method of treatment appears to show that the artificial anus should be left open for at least six months. This obviously in many cases will prevent this treatment, for it is exceptional to find a colotomy plug fit so well

that the patient can carry on her employment. Still, the published cases are on the whole encouraging, and one of our patients appears to be permanently cured. We have found that it is quite unnecessary to wash out the bowel from the artificial to the natural anus. In more than one case the formation of membrane ceased when the bowel was attached to the colotomy wound, even before it was opened, showing apparently a reflex arrest of the morbid process. This quite accords with what we have said as to the importance of the neurotic element in membranous colitis.

Recently Einhorn has adopted the following method of treatment. During the attacks he orders rest in bed, gives some opium, between the attacks he injects eight to fifteen ounces of warm olive oil into the bowel every night. This the patient, if possible, retains. These injections are given nightly for three weeks, and are then gradually reduced in frequency till one is given every week for five or six months.

*Secondary membranous colitis* is not of much clinical importance, for the maladies with which it is associated are so severe as to mask any discomfort due to the membranous colitis. As dyspeptic membranous colitis is rarely fatal, the phrase membranous colitis as used in the dead-house nearly always refers to the secondary form, on the other hand, the phrase as used in practice nearly always refers to the primary variety, for patients affected with the secondary variety do not often pass much membrane.

Direct irritation may cause secondary membranous colitis, as in the cases in which it has followed *mercurial poisoning*, in which condition it is almost certain that the membranous colitis is due to the excretion of mercury into the large intestine. It may also be *septic*, as in the case of a woman who died of puerperal fever, and in whom the whole of the large intestine was buried by a greenish-black membrane. Secondary membranous colitis may also be associated with *Bright's disease*, *pneumonia*, *diabetes*, and *cancer*, and it is said, too, with other specific fevers and tubercle. *Fat necrosis* of the peritoneum has been seen in cases of membranous colitis.

**3 ULCERATIVE COLITIS.**—The colon is frequently ulcerated as a result of typhoid fever, dysentery, tuberculosis, or malignant disease, but from time to time we meet with cases in which this part of the bowel is extensively ulcerated quite apart from any of these diseases. Observation at the bedside has shown that nearly all patients in whom this independent ulceration is found after death have during life presented such a grouping of symptoms as to enable us to predict that the colon would be found ulcerated. To this disease, with its characteristic symptoms and characteristic morbid anatomy, the name simple ulcerative colitis, or, more shortly, ulcerative colitis, is

applied. It is a bad name, because the colon is ulcerated in other diseases, but it is so generally used that much confusion would be caused by any alteration of it at present.

The cause of this disease is unknown, but it is probably due to a micro-organism, and possibly different varieties of it are caused by different but closely allied micro-organisms. It appears to be more common in asylums than in ordinary practice, but otherwise nothing certainly is known of its relationships except that it is often associated with Bright's disease, but it is said in olden days to have been associated in asylums with typhus. Statistics would seem at first sight to show that it has been more commonly met with of late years than formerly, but this is probably due to the fact that we have only recently learned to recognise it.

It is not a disease of childhood nor of old age, for the patients are usually between 25 and 55 years old, it is equally common in men and women.

*Symptoms.*—It is nearly always the state of the bowels which seriously directs a patient's attention to his illness. The first symptom which he remembers is usually abdominal pain, generally gripping, sometimes very sharp and severe, and often sudden in its onset. Soon it disappears, only to reappear later. The duration of these painful attacks varies from a few minutes to many hours, and their alternate appearance and disappearance is very characteristic. The pain is always referred to the front of the abdomen, but its exact position varies in different cases, and also in different attacks in the same patient. Occasionally there is also pain in the back and loins, and once I have known a patient complain of such severe pain in the front and the sides of the chest that she was incorrectly thought to have pleurisy as well as ulcerative colitis. In acute cases the first attack may be excruciatingly severe, but often it does not cause much suffering, the intensity of the pain, however, increases in each succeeding attack. In the interval between the attacks the patient is usually quite free, or he may complain of a dull pain in the abdomen. It should be mentioned that the pain bears no relationship to the ingestion of food, but is commonly worse when the bowels are open. The mere presence of ulcers is insufficient to explain the pain, for it is commonly absent in typhoid fever, it is probably due to some peculiarly irritating ingredient of the contents of the intestines, which stimulates the nerves exposed on the floor of the ulcer, and thus sets up irregular peristaltic contractions. In the majority of cases there is no abdominal tenderness, when this is present it is rarely intense, and most often it is especially marked over some part of the colon.

The presence of severe diarrhoea—sometimes interrupted by short periods of constipation—is almost of equal symptomatic importance to the

pain; it is often the first symptom noticed, and these two symptoms are never absent throughout the whole of a case. The frequency with which the bowels are open commonly varies between two and about a dozen times in the twenty-four hours. Although the act of defecation is often accompanied by abdominal pain, there is rarely the intense tenesmus characteristic of dysentery, nor have I heard of a patient complaining of that almost constant desire to go to stool which is so frequent in a severe dysenteric attack. If the case is mild an occasional solid motion may be passed, but the evacuations are nearly always fluid, dark, foul-smelling, and of a consistency varying between that of slime and water. So far as my experience goes, they never resemble ordinary dysenteric or choleraic stools, indeed I think they are so unlike that the distinction is probably of some diagnostic value. But it is only right to add that some of those whose work lies chiefly in asylums regard the ulcerative colitis they see as a variety of dysentery. Blood is commonly present, some times in considerable quantities, so that the patient describes the motions as being like red currant jelly, sometimes only in traces. It is more often fluid than solid, and is usually bright red, showing that the bleeding has been recent. Commonly it is passed with the motion, although not intimately mixed with it. The clots may be of such a shape as to suggest that they have recently come from the floor of an ulcer.

When diarrhoea is present very little faecal matter is seen in the motions, and what there is consists of a few small lumps scattered about in the fluid. In some cases a little mucus has been observed, but it is never present in large quantities. Often shreddy masses looking very like sloughs are seen, under the microscope they are generally structureless, probably their structure has been destroyed by the action of the contents of the large intestine.

Soon other symptoms are superadded to the pain and diarrhoea. The most frequent of these, and one of the earliest, is vomiting. Often the patients say that they have been vomiting incessantly, but under careful dieting and rest in bed this usually subsides. Occasionally it is entirely absent, it bears no relationship to the intensity of the ulceration. Thirst, probably due in part to the vomiting and diarrhoea, may be very troublesome. Nausea, either with or without vomiting, may be complained of. The tongue is usually covered with a dirty white fur, but as the disease progresses, it becomes red and dry with a brown fur. If the patient be seriously ill, his face has the drawn expression characteristic of abdominal disease. The abdomen is commonly distended, and peristaltic movements may be visible. A rectal examination should never be omitted, for it may be possible to feel the ulceration, and also to determine whether malignant disease is present.

The general symptoms are such as would naturally be expected. The patient gradually becomes wasted, anæmic, and excessively weak, so that he lies in bed looking bloodless, sallow, and extremely ill. The pulse is feeble, and hæmic murmurs may be heard. Irregular pyrexia is often present, the temperature ranging between 100° and 102°, but if the patient be very collapsed it may be subnormal; on the other hand, I have known it as high as 104°. Towards the end of the patient's life he sinks into the typhoid state. The cause of death is usually exhaustion, and in some cases it is distinctly accelerated by hæmorrhage. Occasionally perforation kills. It is noteworthy that, although ulcerative colitis is frequently associated with granular kidneys, suffereis from it do not often show signs of uræmia.

**Prognosis.**—This is very grave—so grave, indeed, that when patients appear to recover from it, it is quite an open question whether the diagnosis was correct, still, sometimes patients with every symptom of ulcerative colitis do get well. The patient is usually dead in eight weeks from the commencement of the illness. The symptoms which especially indicate a speedy termination are great tympanites, much loss of blood, very profuse diarrhoea, and occasionally a high temperature. Some observers state that it is the cause of death of a third of the patients in asylums.

**Diagnosis.**—I think there is little doubt that ulcerative colitis is a distinct disease from dysentery, but the matter cannot be absolutely settled while our knowledge of the bacteriology of the two diseases is so imperfect. Ulcerative colitis does not attack all ages, the bowels are never open 50 or 100 times a day, there is no severe tenesmus, the motions are not dysenteric, vomiting is common, and Bright's disease is often present. In England the two diseases are not likely to be confounded, for acute dysentery is not common, and chronic dysentery is too long-lasting to be confounded with ulcerative colitis. In actual practice the difficulty of diagnosis is usually to separate ulcerative colitis from *malignant disease of the large intestine*, from *intestinal obstruction*, and from some form of *primary anæmia*, but a mistake is usually due rather to a faulty examination of the patient than to any real difficulty.

**Morbid Anatomy.**—Any part of the large intestine may be affected, and generally the ulceration has no special distribution. In an extreme case the muscular coat is exposed, the floor of the ulcers can be seen distinctly to consist of bundles of muscular fibres, and the ulceration is so extensive that only islets of mucous membrane are left here and there, often they are considerably swollen, and consequently they look taller than they otherwise would, and frequently they are more or less stalked because of the ulceration which undermines them. The

result of this is that a careless observer takes the islets of mucous membrane for polypoid growths, and the exposed muscular coat for the natural level of the colon. The vessels of the mucous membrane are dilated. The number of ulcers varies from one or two to several dozens, but while ulcers of recent date are often more or less circular, and vary in size from a pea to a five-shilling piece, by the time death occurs their shape is usually very irregular, and they have run one into another. Attempts at repair are very rare. Perforations may be very numerous. In some cases the small intestine is affected as well as the colon, but this is rare, and the process is much more severe and apparently of longer standing in the colon. The liver is often fatty.

*Associated Diseases*—I have already mentioned that the disease is common in asylums, and there patients dying from ulcerative colitis are frequently found to have pneumonia. Gemmel thinks the onset of pneumonia always fatal. Outside asylums, chronic Bright's disease is the condition most frequently associated with ulcerative colitis, and even in Gemmel's asylum, out of eighty fatal cases of ulcerative colitis the kidneys were cirrhotic in thirty-five. One of Bright's original cases of albuminous urine is that of a woman aged 40, in whom the kidneys were hard, tough, and lobulated, and the large intestine was ulcerated throughout. Pyæmic hepatic abscesses are very rare, but occasionally a single large hepatic abscess occurs in association with ulcerative colitis.

*Treatment*—Unfortunately this avails but little. The patient must remain in bed and be kept warm. If the pain be very severe, hot abdominal fomentations are comforting. The diet should consist of milk, and often a little brandy is required. Bearing in mind the abdominal pain, and that the diarrhoea is often excessive, opium is probably the best drug to prescribe. In a severe case the patient should be kept well under its influence, it matters little what preparation is given, care of course being exercised if the patient has chronic Bright's disease. Half or three-quarters of a pint of a saturated solution of boracic acid at about 100° F may twice a day be let to run slowly into the bowel through a long rectal tube introduced as far as possible while the hips are raised. Gemmel uses salol dissolved in turpentine in the same way, and he attaches much importance to the administration of quinine by the mouth. The administration of *Ol. encalyptus*, in capsule form (*m v t i d*), has been found of service, and deserves a further trial. Perhaps in suitable cases it would be a good thing to open the colon on the right side, to let the fæces be discharged through the artificial anus, and then to syringe through from it with some antiseptic solution.

**OTHER VARIETIES**—*Follicular ulceration* is of very little clinical importance, so it need not

detain us long. It begins by an accumulation of small round cells in the solitary follicles, which therefore swell, later they rupture and the ulcers are formed. These ulcers do not extend deeply, they have sharply cut edges, and in a well-marked example the gut is quite honey-combed with them, varying in size from a hemp-seed to a large pea. I have never met with any case in which there was an attempt at repair, nor, on the other hand, have I ever heard of perforation, for the floor of the ulcer is always formed by the muscular coat.

The patients in whom follicular ulceration is found have always died of some other disease, often of the gastro-intestinal tract. Thus I find that of ten adults, one had dysentery, one cancer of the rectum, one membranous colitis, one typhoid fever, and one femoral hernia. Follicular ulceration of the colon occurs about once in every 500 post-mortems in hospital practice. It is never diagnosed during life, for the symptoms of the fatal disease quite overshadow any that might be attributable to the ulceration. It is proportionately more frequent in children than in adults. Thus Holt met with follicular ulcers in the intestine in twenty out of seventy fatal cases of infantile non-tuberculous diarrhoea. They were never seen in cases that had lasted less than a week, and the proportion of follicular ulceration was highest in those that had lasted more than ten weeks. When in the small intestine they were always most numerous near the cæcum.

*Vascular ulceration* of the colon is chiefly of pathological interest. There are two varieties of it—venous and arterial. The venous form is by no means uncommon, and is probably due to the same cause as venous ulceration elsewhere. The arterial is due either to blocking of one of the mesenteric arteries by an embolus, to arterial thrombosis, or to atheroma, especially that of the aorta just at the origin of the mesenteric arteries. None of these are sufficiently common to call for notice here.

*Hæmorrhagic Ulceration*—In some diseases submucous extravasations of blood take place into the colon, and these no doubt may break down and lead to ulceration. Hæmorrhagic ulcers are usually small, shallow, rounded, and discrete, although occasionally two or three may run together, submucous hæmorrhages may commonly be seen in their neighbourhood. Hæmorrhagic ulcers are by no means uncommon in those who have had Bright's disease. This is what might be expected, for we know that persons with Bright's disease are particularly liable to hæmorrhage from any part of the body. They may occur too in other diseases, such as purpura, scurvy, and other forms of anæmia.

*Trophic Ulceration*—It is by no means proved that the intestine is ever ulcerated as a result of lesions of the central nervous system, but several cases have been put upon record—and quite

recently I have seen one not yet recorded—in which intestinal ulceration was associated with disease of the spinal cord. The following is an instance: A man had complete paraplegia as a result of a fracture of the spine in the lumbar region. There were numerous small round ulcers occupying the whole of the colon. Many more cases will have to be observed before the question of trophic intestinal ulceration can be settled, but as a lesion of the spinal cord can lead to an ulcer of the lower extremities, it is not unlikely that it might lead to an ulceration of the intestine.

**DILATATION OF THE COLON**—Cases in which the large intestine is dilated may be divided into four groups.

The *first* contains those in which the distension is entirely gaseous, is not due to any obstruction, and is only one symptom of some other illness. It is often seen in association with peritonitis and typhoid fever, and may occur in almost any severe illness. When extreme it is of great importance, for it adds considerably to the danger of the original disease, and greatly hampers the movement of the heart and lungs. The abdomen is very distended and tense, it hardly moves on respiration, it is hyperresonant on percussion, and the tympanic note extends well into the flanks, the liver is pushed up and the splenic dullness is obliterated, the patients are nearly always constipated and do not pass much flatus per rectum, but they complain of borborygmi which may be audible to bystanders. The gas consists chiefly of carbonic acid and hydrogen. The only difficulty of diagnosis likely to occur is in the distinction of this condition from those rare cases in which there is gas in the peritoneal cavity.

The treatment of tympanites is very difficult and uncertain. Various carminatives, especially the aromatic oils, are often recommended, but although I have often given them I have never seen them to be of any use in serious gaseous distension of the large intestine. Enemas give a better chance of success, those of asafoetida and turpentine are the most useful, but we naturally shrink from the use of large enemata in typhoid fever or peritonitis. Puncture of the bowel has been recommended. It should be done with a sterilised trocar and cannula, but it often fails to give relief, for the gas soon collects again. Acupuncture should never be employed, for it often leads to an escape of gas into the peritoneal cavity.

The *second group* contains those cases in which dilatation of the colon is due to some solid substance within it. Concretions consisting of vegetable fibres, hair, and other foreign bodies are sometimes found in the insane. A gall-stone may rarely block the colon, but in the immense majority of cases in which it causes intestinal obstruction it stops in the lower part of the ileum. Distension of the large intestine by

fecal matter is of great importance, and fecal impaction is a common cause of intestinal obstruction, under which heading it will be found described.

The *third group* contains those cases in which the dilatation of the colon is due to some organic obstruction in front of the dilated duct. The muscular coat of the dilated gut usually undergoes hypertrophy. The intestinal contents retained behind the obstruction set up some colitis, which may proceed to ulceration, and so the well-known distension ulcers are produced. These may be very numerous. For example, one case is described thus: "The whole of the colon above the stricture was distended and worn-eaten by small ulcers." Sometimes this leads to perforation and consequent peritonitis. It is curious that occasionally the ulcers may be a long way behind the obstruction. This colitis may be difficult to treat after the obstruction has been overcome. The dilatation behind an obstruction may be very great and lead to much abdominal distension. By careful watching of the peristaltic movements, by percussion, and by palpation we can often make out whether it is large or small intestine that is dilated, but it must not be forgotten that a distended large intestine may be bent down towards the pubes like small intestine, or a distended small intestine may be as large as a normal large intestine.

The *fourth group* contains those very rare cases of so-called idiopathic dilatation of the colon. A few cases which have been recorded as examples of this condition are really instances of fecal impaction leading to great dilatation of the colon behind the impacted mass. Putting aside these cases, a well-defined group exists among those of *idiopathic dilatation of the colon*, and the characteristic of this group is that the dilatation is of the sigmoid flexure. It may be enormous, filling nearly the whole abdomen, displacing the liver upwards, and resembling either an enormously dilated stomach, or so bent on itself as to form two huge sacs lying vertically side by side in the abdomen. It may measure two feet in circumference. When the dilatation is not of the sigmoid flexure it is usually near it. It has been suggested that the dilatation in all these cases is due to kinking, set up by the presence of impacted feces, but this suggestion is probably not correct, for many of these patients do not give a history of constipation, fecal impaction is very common, but the condition under discussion is excessively rare, and, lastly, often at the autopsy the distended bowel was not full of feces. Probably *idiopathic dilatation of the colon* is allied to those cases of dilated stomach in which there is no obstruction at the pylorus, and this is especially likely to be true of those cases in which the dilatation is confined to the sigmoid flexure.

The symptoms of idiopathic dilatation of the colon can be very briefly described, the condition



is much commoner in males than in females sometimes occurs in young children, having in them probably existed since birth. The distension of the intestine is very great, and percussion shows that it is chiefly due to gas. Shortness of breath is a cause of complaint, and the patient may be livid because the diaphragm is pushed up and the breathing hampered. The upward displacement of the heart gives rise to palpitation. The splenic dulness is obliterated, the hepatic dulness is diminished, and the liver cannot be felt. The abdomen is always distended, sometimes enormously so, and then it may be tympanitic all over, usually neither coils nor peristaltic movements are visible. In very rare cases the pressure has been so great as to lead to oedema of the legs, scrotum, and penis, and to albuminuria. Most of the patients for some time, often for years, have had some difficulty with the bowels, constipation supervening from time to time, but this is not usually very serious, and has yielded to purgatives or enemata. After death, both layers of the muscular coat are found to be hypertrophied, and there is often some ulceration of the dilated mucous membrane. The distended bowel contains some semi-solid faeces and an enormous quantity of gas. Of the recorded cases all the patients except one have died. Occasionally death is due to perforation of an ulcer, but more frequently the cause of it is by no means clear, and then it may be sudden.

The treatment has hitherto been very unsatisfactory. Probably the best course would be to open the bowel above the dilatation, wash out the dilated part, and let the faeces pass for some time through the artificial anus, and so give the affected part rest.

**SACCUATION OF THE COLON** is not a common condition. The diverticula are usually the size of a pea, but they may be large enough to receive the little finger, and half an inch in depth. They are most frequent in the descending colon, sigmoid flexure, or upper part of the rectum. Usually they are very numerous, are placed close together, and contain a little faecal matter. They are to be regarded as hernial protrusions of the mucous membrane through the muscular coat. They are found for the most part in elderly people. Only once have I heard of their giving rise to symptoms, and that was in a case in which the perforation of one of these sacculi led to the formation of an abscess between the left kidney, spleen, and ascending colon.

**INJURIES OF THE COLON**—*Ulceration of the Colon*—Sometimes, but very rarely, this is due to an enema tube or bougie, but it is not very infrequent for a nurse or a doctor to be excessively afraid that the administration of an enema has led to perforation of the bowel, for in elderly people the evacuation of the bowel by an enema is often accompanied by much faintness and even by syncope. The pulse may become quite small and the patient appear

collapsed. Elderly or feeble people should never be allowed to get out of bed for the evacuation of the bowels after the administration of an enema unless they are in the habit of using enemata.

The kinds of accident that most frequently damage the intestine are being run over, kicked in the abdomen, or stabbed there. The small intestine is much more often affected than the large. (See "Abdomen, Injuries of.")

**Perforation of the Colon from without**—All sorts of abscesses may burst into the colon, thus iliac and psoas abscesses, localised peritoneal collections of pus, hepatic abscesses, appendicular abscesses, splenic abscesses, ovarian abscesses, suppurating growths, abscesses of the gall-bladder, and hydatids of the liver may all burst into the colon. This vent is usually recognised by the fact that the original tumour becomes smaller, and there is a discharge of pus by the bowel.

**Malformations of the Colon**—These are excessively rare, but they depend for the most part upon malposition of the caecum. It will be remembered that the first position of this structure is outside the abdomen, then inside just under the umbilicus, then to the lower part of the left side of the abdomen, next near the cardiac end of the stomach, later under the liver, and lastly in the right iliac fossa. The caecum may remain in any of these positions, while the colon continues to grow. This naturally gives rise to various malformations.

**METALLIC PIGMENTATION OF THE COLON**—Sometimes lead may be deposited in the tissues of the colon. Thus a man who had lead colic died from chronic Bright's disease. The whole of the caecum and large intestine was very black. The colon contained 0.086 per cent of lead. In this case as in others the pigmentation at the caecum began very abruptly. There is nothing to show that lead is not deposited in the colon by the same process as leads to the formation of a blue line on the gums.

In very rare instances mercury and bismuth may cause black pigmentation of the colon.

**MALIGNANT DISEASE OF THE COLON**—A primary growth in the colon is almost always a cylindrical celled epithelioma, it is usually circumscribed to a small spot, and then by its contraction sets up intestinal obstruction. Occasionally, however, the growth is soft and medullary, then it breaks down in the centre, and intestinal obstruction does not arise. Thus there are clinically two groups of cases of malignant disease of the colon—those without and those with symptoms of intestinal obstruction.

A post-mortem examination on the group of cases in which there is no intestinal obstruction reveals that several inches of colon are affected with growth, usually forming an obvious tumour visible directly the abdomen is opened. On cutting into this mass it is found that the

normal channel of the intestine is replaced by an elongated cavity with black, ragged, sloughy walls, but passing into healthy colon above and below. I have known the whole of the transverse colon to be converted into such a mass. The contents of the cavity consist of a foul mixture of faeces, blood, and necrotic growth. If the sigmoid or caecum is affected this mass may grow into the subjacent bone, if it is in other parts of the colon, it may extend into the stomach, liver, or kidneys. This state of things during life may give rise to no symptoms, on the other hand it may be easy during life to detect a tumour, especially if it is in the transverse colon or the caecum. The patient may pass large quantities of foul, dark grey, or black fluid motions, in which, perhaps, fragments of growth and small blood-clots are seen. Sometimes between the passage of such motions the bowels may be constipated. The patient suffers pain, he wastes, and may even die before any secondary deposits show themselves. When the tumour is in the caecum or the sigmoid, it may cause oedema of one leg and thrombosis of the external iliac vein, and when it is in the transverse colon, we must carefully distinguish it from an enlarged liver, carcinoma of the greater curvature of the stomach, or an omentum puckered up by some form of chronic peritonitis. The implication of other organs by direct spread of the growth rarely gives rise to any symptoms by which it can be recognised. In very rare cases of carcinoma of the large intestine a fistulous tract is formed, which opens either into the stomach, the duodenum, or some adjacent coil of small intestine. In rare instances the carcinomatous growth ulcerates directly into the peritoneal cavity.

We now pass on to the description of the second group of cases of malignant disease of the colon, namely, those in which the growth produces obstruction. This variety of growth is usually limited to a quite small portion of the bowel, and forms an annular band, encircling the whole circumference of the affected part of intestine. The gut from the outside appears suddenly constricted, as though some one had drawn a tight string around it. The stricture is therefore quite narrow, and the peritoneal covering of the gut is usually thickened just over the stricture. From the inside the stricture appears as an annular and contracted deposit, the surface of which is irregularly ulcerated. The stricture may be extremely narrow, so that often the wonder is that the patient has presented so few symptoms. The effects that occur behind the stricture of the bowel have already been described, they are dilatation with hypertrophy of the colon and distension ulcers. The presence of these makes life hazardous. I remember a case in which a surgeon was to have operated on a patient one evening, but the operation was put off until the next morning, and in the

night the patient died from perforation of a distension ulcer. On the other hand, in very rare cases the opening of a distension ulcer into some adjacent viscus, *e.g.* the bladder, has relieved the symptoms of obstruction, or an abscess may form around the distension ulcer, this abscess bursts externally, and thus a faecal fistula is formed.

With regard to the locality of stricture of the colon, Treves gives the following table of 100 cases —

Sigmoid flexure	58
Descending colon	11
Splenic flexure	7
Transverse colon	7
Hepatic flexure	9
Ascending colon	2
Caecum	6
	100

The symptoms of malignant stricture of the large intestine are as follow — The patient, who is usually a man of over fifty, says that for some time past he has been losing flesh, and that he has been liable to attacks of abdominal pain, not always severe, and, to begin with, only coming on at long intervals. The attacks get more and more frequent, and gradually there may be constant pain with exacerbations. Concurrent with these symptoms difficulty of keeping the bowels regular appears. There are attacks of constipation which are at first easily relieved by aperients, but as time goes on the patient has to take more and more aperients, until at last he comes to a doctor because nothing that he takes will unlock the bowels, and then he begins to suffer from abdominal distension, nausea, with sometimes actual vomiting, loss of appetite, and furred tongue. But this by no means completes the picture, for one of the most striking things about malignant disease of the large bowel is that these attacks of constipation often alternate with attacks of diarrhoea. This is due to the fact that the retention of faeces above the stricture sets up a colitis. Thus it follows that irregularity of the bowels is one of the striking features of this disease. The motions are often very characteristic. Thus when solid they may be altered in shape and also of small circumference, owing to having passed through the stricture, occasionally the patient describes them as tape-like or pipe-like. Too much stress is, however, usually laid upon the shape of the motions, for it must be remembered that their shape is for the most part acquired in the rectum. But when diarrhoea is present, the motions are commonly foul-smelling, contain mucus, and it may be, necrotic fragments of growth. Whether there is constipation or whether diarrhoea is present, blood is often seen in the motions. The rectum is often stated to be ballooned, that is to say, when the finger is

introduced through the anus, the rectum is found to be empty, and so dilated that the finger can scarcely reach the walls, which are perfectly smooth. The cause of this condition is quite unknown, it is not always present in malignant disease of the colon, and it may be met with in other conditions. The abdomen is often very distended, and visible coils of intestine may be seen contracting through the abdominal parietes. These are often more easily visible the longer the case, for hypertrophy of the muscular coat of the distended bowel renders them especially conspicuous, but it is by no means necessary that the bowels should be hypertrophied for them to be visible, for I have seen them well marked when obstruction had only existed twenty-four hours. Sometimes a tumour can be felt through the abdominal parietes. This is more often fecal accumulation than growth. If the growth extends low down enough it may be possible to feel it per rectum, and it may be that attention is directed to the fact that the bowel cannot hold a copious enema. Frequent rumbling and gurgling signs are heard in the abdomen. In the group of cases now being described, intestinal obstruction occurs sooner or later, and it is important to remember that even in a chronic condition, such as malignant disease of the colon, the signs of intestinal obstruction may come on acutely, and as, further, the signs of malignant disease of the colon may be unobtrusive until acute obstruction sets in, serious mistakes in diagnosis are by no means uncommonly made. Thus I have seen a case which was really one of acute intestinal obstruction due to malignant disease of the colon called acute peritonitis, but as a rule the intestinal obstruction is chronic, and the patients give a clear history of malignant disease of the colon. Sarcomata and innocent tumours of the colon are too rare to call for mention here.

**Colonisation.**—The treatment of the insane by their collection together in a village under superintendence but without confinement, the family or cottage system of provision for the insane, such a colony exists at (heel

**Colopexia.**—The fixing of the sigmoid flexure by sutures to the abdominal wall, if this part of the colon has been incised before the suturing, the operation is called *colopexotomy*.

**Coloptosis.**—Downward displacement of the colon. See ENTEROPTOSIS.

**Colorado.** See THERAPEUTICS, HEALTH RESORTS (*American*).

**Colorimeter.**—An apparatus for determining the quantity or intensity of colour in anything, e.g. in the blood. See BLOOD (*Estimation of Haemoglobin, Hoppe-Seyler's Colorimetric Method*).

**Colostomy.**—An operation by which a permanent opening (to serve as an artificial anus) is made into the colon, in *colo colostomy* two parts of the colon are connected together by making an opening.

**Colostrum.**—The first milk that comes from the mammary glands after the birth of the child, the "beestings" or "green milk." *Colostration* is a disease of new-born infants supposed to be due to the colostrum. See INFANT FEEDING (*Human Milk, Colostrum*), PUERPERIUM, PHYSIOLOGY (*Lactation*).

### Colotomy.

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See also COLON, DISEASES OF, COLOSTOMY, INTESTINES, SURGICAL AFFECTIONS OF, etc.

THE operation of colotomy, or the formation of an artificial anus, is undertaken for certain grave maladies of the rectum and of other parts of the large intestine, and the modes of executing the operation, and the place at which it is to be performed, depend themselves on the nature and the degree of gravity of the ailment. Formerly colotomy was regarded as an operation to be postponed until obstruction was complete, now it is more generally used, it should not, however, be employed without due consideration.

INDICATIONS FOR.—Cancer of the rectum and of other portions of the large intestine, and strictures and ulceration, whether tuberculous, syphilitic, or dysenteric, etc., sometimes combined with extensive fistulae, are the main diseases that call for colotomy, provided that intestinal anastomosis or resection of the stricture is found to be impossible. The chief conditions, besides obstruction, which may necessitate colotomy, are great pain, severe hæmorrhage, and intense and persistent diarrhoea.

When the growth or stricture causing the obstruction is within the rectum its exact position can be felt, and the surgeon can determine whether to adopt other means or to resort to colotomy, but when the obstruction is in any other portion of the large intestine, unless a growth can be definitely discovered, it is wise to make an abdominal exploration, so as to find out the position of the obstruction and the best mode of dealing with it.

In some forms of cancer of the rectum and other forms of ulceration the great pain caused by faeces passing over the growth or into fistulae about the buttocks of itself necessitates colotomy.

The severe and dangerous hæmorrhage caused by the passage of feces over very vascular growths may sometimes demand colotomy.

Colotomy is frequently required for persistent diarrhoea arising from cancer of the lower part of the sigmoid flexure and of the upper part of the rectum, or from syphilitic or tuberculous ulceration, not only of the lower, but also of the upper parts of the large intestine. In such ulcerations, tuberculous, syphilitic, or dysenteric, colotomy, performed above the diseased portion of the gut, will stay the diarrhoea.

Colotomy falls under three heads, namely, *inguinal colotomy*, left or right, *lumbar colotomy*, left or right, and *transverse colotomy*. The left *inguinal* and the left *lumbar* operations are far the most frequent, and a general preference may be given to the former. The opening being in front in inguinal colotomy, it can be attended to by the patient much more easily than is the case in the lumbar region, and a pad or tuess can be more readily adjusted. The importance and possibility of being able to procure a good spur, and so preventing feces from passing below the colotomy opening, are strong points in favour of the inguinal method. Especially is this the case if the colotomy has been done for the relief of pain, hæmorrhage, or diarrhoea.

Again, a spur can always be made in the inguinal region, but this is not so in the lumbar region, as often the gut is too firmly fixed to the loin, and at too great a depth from the surface wound.

For transverse, right lumbar, and right inguinal colotomy the rule is to perform the colotomy immediately above the stricture, or stricture with ulceration, if such be clearly indicated. In uncertain cases a median abdominal exploration should first be made, and when the seat of the obstruction is found, the colotomy should be done above that, thus for disease about the splenic flexure of the colon, transverse colotomy is the best, for disease at or extending up to the hepatic flexure, right lumbar colotomy is preferable, for disease lower down, right inguinal colotomy should be chosen. If the exploratory operation has not had definite results, right inguinal colotomy is the best, so that the surgeon may work well above the diseased part of the large intestine.

#### OPERATIONS

(a) *Left Inguinal Colotomy*.—In left inguinal colotomy, about an inch and a half inside the left anterior superior spine of the ilium, and parallel with Poupart's ligament, the skin and cellular tissue are divided by an incision not more than two inches in length, and often less. The muscles are divided down to the subserous areolar tissue, which is torn through, the peritoneum is opened, the finger is introduced into the opening, and the deep structures are

divided with scissors up to the extent of the skin wound.

My former practice was to stitch the parietal peritoneum to the skin, now I think it wiser not to do this, as it was shown by the late Dr. Greig Smith that a much better union is obtained when the peritoneum covering the gut unites to muscles and cellular tissue than when peritoneum becomes united to peritoneum.

The gut is sought for and brought to the surface, and is at once recognised to be large intestine by its longitudinal bands and appendices epiploicæ. While it is held out of the wound the mesentery fixing it to the back of the abdomen is sought for. Then through the mesentery, just as it joins the gut, a pair of Spencer Wells's forceps (closed) are passed, and these forceps, perforating the mesentery, are allowed to rest across the wound in the abdominal wall. They thus support the gut, and prevent it from slipping back again into the abdomen. The handle of the forceps should point toward the middle line, the apex or point of the forceps should point outwards.

A suture is then passed through the serous and muscular coats of the bowel, and through the skin at the upper and lower angle of the skin wound. If necessary, three or four other sutures may be used in like manner to unite the gut to the skin about other parts of the wound.

I now use forceps rather than a glass rod or the mesenteric stitch, as they are not so likely to slip out, as may happen when the glass rod is used, or tear through prematurely, as is at times the case with the mesenteric stitch.

It is most important that these forceps should be kept through the mesentery behind the gut for at least ten days, after which they are removed.

In from twenty-four hours to three days after the operation, the gut is opened by a transverse incision over the forceps and parallel to them. It should be made only long enough so as to allow flatus or liquid feces to pass out.

In about ten days' time, or longer, if the union between the gut and the wound does not appear to be very strong, the entire gut is cut through in a transverse direction, the incision going through the opening made to let out flatus and feces. This cut is carried down to the clip forceps through the mesentery—in fact, it absolutely divides the gut across, and thus frees the forceps. By cutting down on to the forceps, one can make sure that the gut is really divided in two, for, as previously shown, the forceps at the original operation are passed through the mesentery behind the gut.

By the cutting of the gut transversely there is no pain, and little or no bleeding, as the nerves and vessels run transversely round the gut.

The essential point of the operation is to prevent feces passing below the artificial inguinal opening, and to effect this the gut is fixed up by the forceps through the mesentery

—in other words, a spur is procured. Unless this is obtained there will not be an artificial anus, but a fecal fistula, beyond which feces pass into the distal portion of the gut. Thus the fecal irritation is continued, and the operation has failed in one of its objects. But when a proper spur is made, feces pass through the inguinal opening only, and cannot enter the distal end of the intestine.

(b) *Right inguinal colotomy* is performed by the same incision as on the left side, but this incision should be lower down and nearer to Poupart's ligament. The cæcum is the region to be opened. The question of a spur can never arise.

In cases demanding this operation, when the cæcum is very distended, Paul's tube is of very great service. The peritoneal cavity is opened, and the cæcum is drawn into the wound and incised, a small Paul's tube is at once inserted, and the cæcum tied around it. To the free end of the glass tube a drainage tube is attached to carry the feces away to some vessel remote from the wound. More stitches are used to fix the cæcum to the edge of the wound. The Paul's tube is removed in a few days, when the cæcum is well glued up to the surface. Paul's tube should be used in all parts of the large intestine when distension is great and it is necessary to open the gut at once.

(c) *Lumbar colotomy* can be performed in several ways, the direction of the incision and the mode of fixing up the gut being the main subjects of difference.

The best incision for finding the colon is one with its centre half an inch posterior and midway between the anterior superior and posterior superior spines of the ilium, and midway between the last rib and the crest of the ilium. From this point Callison used a vertical incision, but the length of this is limited, and it is not easy to work down upon the gut. More room is given in difficult cases by Amussat's transverse, and Bryant's oblique incision.

Half an inch behind the point described, and with its centre over the chosen spot, an incision (either transverse or oblique) is made, not longer than two inches, for otherwise the gut may be missed. The skin and cellular tissue are divided, and the muscles exposed and quickly divided till the fascia lumborum is reached. This is opened, and the quadratus lumborum is exposed at its anterior edge. The edges of the wound are retracted, and the fat around the kidney and the fascia lumborum is opened up. Then the gut will bulge into the wound, if it is distended and has no mesentery. In straightforward cases it will be recognised to be the colon by its being uncovered by the peritoneum, for if the peritoneum is opened, peritoneum will be seen surrounding the gut, and the longitudinal bands, and the appendices with these, will also be visible. The colon is then brought to the surface, and stitched all round to the skin wound with inter-

rupted sutures, which should pierce the muscular coat only, and not perforate the gut.

But rarely is this actual presence of the colon made absolutely certain without opening the peritoneum, and the difficulties arise from the position of the intestine in relation to its peritoneal covering and length of mesentery. In what was formerly, but erroneously, supposed to be the general position, the peritoneum covers only half or two-thirds of the circumference of the gut, leaving the posterior part uncovered, with the intestine bound down to the loin. If no longitudinal band is seen, there is a danger of opening the small intestine, or even the stomach, imagining that the large intestine is being dealt with. I am therefore strongly of opinion that one should intentionally make a small incision in the peritoneum towards the inner part of the wound, and, by finding intestine with longitudinal bands or appendices epiploicæ, become certain that the large intestine has been reached.

In a second condition, where the colon is entirely surrounded by peritoneum and has a mesentery, it cannot be reached, nor can the longitudinal bands be seen, without first opening the peritoneal cavity. By the index finger inserted into the peritoneal cavity the gut can be felt and hooked up and sutured, as above described, into the loin wound.

In a third condition the mesentery is very long, and the intestine may alter its position in the abdomen so as to be on the side opposite to that in which the incision is made. Prior investigations failing, the external wound, and the peritoneum to a corresponding extent, must be enlarged so as to admit the hand, and a search is made for the splenic or for the hepatic flexure of the colon. From one of these points the colon can be found, and brought to and fixed to the skin wound.

Right lumbar colotomy is performed in the same way and with the same difficulties, as the mesentery is usually fairly long, and therefore it is most important to open the parietal peritoneum to make sure that the large intestine is being dealt with.

(d) In the infrequent operation of *transverse colotomy* the rectus muscle just above the umbilicus is exposed and the posterior part of its sheath incised, and the peritoneum is picked up and divided, and then, as in inguinal colotomy, the large intestine must be found and recognised by its longitudinal bands, and then pulled forward and fixed outside the abdomen. The gut is secured to the skin by sutures through the peritoneal and muscular coats. The incision should always be large enough to admit the introduction of the hand into the abdomen, to make sure that the colotomy is being done above the ulceration or stricture. Then the lower part of the wound is brought together as in abdominal section, the upper two inches are treated as in inguinal colotomy, and through

them the transverse colon is brought and fixed. When a spur is required to prevent faeces passing beyond the opening, a clip can be put behind the gut as in the inguinal operation.

**AFTER-TREATMENT.**—For all the forms of colotomy the after-treatment, *mutatis mutandis*, is the same. After the operation the patient is put back to bed, lying on the back, with the head low and a pillow placed between the knees. The use of opiates is dissuaded, unless necessitated by very great pain, restlessness, or severe purging. For the first twenty-four hours as little food as possible is given, the preference being for nutrient enemata, when possible, or soda and milk, weak tea for washing out the mouth, and brandy and water in rare cases of faintness.

Several points should be noticed on the first visit on the evening after the operation. Pain in the back may be remedied by turning the patient on the side opposite to that on which the colotomy has been performed, not on the same side. Any probable bronchitis may be prevented by the patient being propped up in bed. If wind causes much distension or tympanitis, the bandages should be loosened, or, if necessary, the dressings may be removed and the gut slightly punctured with a lancet, so that flatus may escape. The question of the passage of urine should be soon to.

The next day the dressings are left undisturbed, and the diet is still kept meagre and liquid, beef-tea being permissible.

The second day after the operation the dressings are removed, and the gut is opened by scissors for about one or one and a half inches in a transverse direction of the gut to allow of the escape of wind or faeces. This may be done earlier or deferred, according to circumstances. Any small vessels that bleed are clipped or ligatured, slight oozing of blood is allowed to clot, absorbent wool is applied to the wound, and dry gauze is used as a covering. After this, diet can be fruer, fish being allowed first.

The day after the gut has been opened the bowels should be made to act, castor oil or liquorice powder being administered. Any impaction or hard faeces that block the way should be carefully broken up by the finger and olive oil injected. The wound is dressed with wool soaked in any antiseptic. Thenceforward the bowels must be carefully kept open.

In ten days' time, when the bowels have been well emptied, the gut should be cut across as shown in the description of the inguinal operation.

A little later the patient may rise from bed and lie on a sofa, with the colotomy opening dressed with ointment and supported by a pad of wool. Any tendency to contraction about the proximal opening of inguinal colotomy is corrected by the daily passage of the finger into the upper opening, the lower one being allowed

to contract as much as possible. Old faeces lodged in the lower bowel may cause rectal irritation. These should be disposed of by irrigating with water into the lower of the two orifices towards the rectum, and then from the rectum through the lower orifice. In a short time the lower portion of the gut will contract and become merely a passive tube.

In about three weeks the patient can walk, wearing either a truss or abdominal bandage. The bowels should be made to act in the morning, a kidney-shaped bowl being placed under the colotomy opening. After left lumbar or left inguinal colotomy the motions are usually quite solid. After transverse, right lumbar, or right inguinal colotomy, through which the action of most of the large intestine is stopped, the colotomy opening being so close to the small intestine, the faeces are consequently liquid, or semi-liquid. This after-result tells against the three higher operations, and points to the advisability of doing the colotomy as low down the large intestine as is consonant with the position of the disease.

**Colour-Blindness.** See AMBLYOPIA (*Hysterical*), COLOUR VISION (*Conjugal and Acquired Colour-Blindness*), Hysteria (*Sensory Disorders, Ocular Anesthesia*)

### Colour Vision.

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See also BRAIN, PHYSIOLOGY OF (*Sensory Centres, Half-Vision Centre*), PHYSIOLOGY, THE SENSES (*Vision, Colour*), PHYSIOLOGY, THE SENSES (*Vision, Colour Sensation, Complementary Colours, etc.*), SPECTROSCOPE IN MEDICINE (*Colour*)

**PHYSIOLOGY OF NORMAL COLOUR VISION.**—The complex subject of "colour vision" lies on the borderland between the realms of Physics and Physiology, and an intelligent appreciation of its many problems demands an acquaintance with both the physical and physiological sides of the question.

**Physical Considerations.**—The various physi-

cal forces in nature which emanate directly or indirectly from the sun are regarded by modern physicists as the expression or outcome of undulations or wave-like motions propagated in the hypothetical all-pervading medium—the ether. These undulations vary considerably in length and frequency, and in the associated physical effects which they produce upon animate and inanimate nature.

Ascending the scale of ethereal disturbance from the long waves of comparatively slow oscillation to the shortest and most rapid undulations, modern physicists assume the existence (in sequence) of ethereal rays possessing electromagnetic, thermic, photogenic, and actinic or chemical properties. The first and last groups of this series appear (in the present state of our knowledge) to be devoid of effect on the human organism, or, at least, we do not possess any specialised receptive senso-organ conferring on us a conscious knowledge of their existence. The highly specialised nerve-endings of the skin and retina, however, provide the appropriate receptive channels for those physical stimuli which, by some peculiar and unknown transformation therein, endow us with the faculty for the conscious perception of heat and light. At present we are only concerned with the photogenic ethereal rays, in so far as a consideration of them is essential to the comprehension of the physiology of normal colour vision.

As above premised, the sun is the great source of natural light in our universe, and the whole of the photogenic rays thence emitted are collectively appreciated by us as the solar white light. This apparently simple light is, however, of a composite character, for by means of the spectroscope it can be analysed into several component and further indivisible parts, constituting the defined colours of the spectrum with the multitudinous variety of hue or colour-tone resulting from their gradual transition into each other. In the light of the now universally accepted undulatory theory, this phenomenon of spectral colour-dispersion depends upon the constitution of the solar white light, which is composed of many groups of ethereal rays corresponding to the individual colours, and varying in their vibratory rate and wave-length, and accordingly in their refrangibility by refractive media. Colour, therefore, is the physical equivalent of wave-length and frequency, and in accordance with these attributes, the related rays produce correspondingly different impressions upon the receptive retina, which the interpretative mind appreciates as variation in colour.

It has been experimentally determined that the range of our appreciation for photogenic waves begins when the ethereal vibrations number about four hundred billions per second, and the wave-length is about  $750 \mu\mu^1$  (which is

equivalent to the sensation of red), and continues through the series of increasing vibrations (represented by orange, yellow, green, blue, and indigo) until the rate of oscillation has increased to about eight hundred billions per second, and the wave-length has inversely diminished to about  $380 \mu\mu$ , when the sensation of violet is produced. Beyond this point their perception as light ceases, and they manifest their existence by the possession of actinic or chemical properties.

Upon this compound character of the solar light the varied phenomena of colour depend. Colour is not (as was formerly supposed) an inherent quality of the objects around us, but is a purely subjective sensation produced in the special perceptive brain centres of animate creation. The infinite variety of colour seen in external objects depends upon the selective power which such objects possess of absorbing and quenching, in varying degrees, certain of the component spectral colours of the composite light which illuminates them, and reflecting the remainder to the perceptive sense organ.

Associated with every colour sensation the existence of certain distinctive attributes, known as colour constants or elements, can always be distinguished. These are three in number: (1) *hue*, (2) *purity* or *tint*, and (3) *brightness*, *luminosity*, or *shade*.

*Hue* is synonymous with ethereal wave-length, for upon this physical factor colour is solely dependent.

*Purity* or *tint* depends upon the absence of white light, the less this admixture the purer the colour.

*Brightness*, *luminosity*, or *shade* depends objectively upon the energy of vibrations of the ether particles, and subjectively upon the sensitiveness of our retina to this photogenic physical stimulus.

By means of these varying characteristics or qualities any given colour can be scientifically defined in terms of the constant spectral colours.

*Physiological Considerations*—The delicate and complex nervous expansion—the retina—is destined for the amplification and support of the ultimate terminal elements of the optic nerve fibrils—the rods and cones. The phenomena of Purkinje's figures and the blind spot establish the fact that these are the essential visual cells, to which (through the inner layers of the retina) the photogenic ethereal waves must first penetrate before visual impulses can originate.

Upon these nerve terminals the ethereal impulses of from four to eight hundred billions per second—the physical equivalent of their respective colour-sensations—impinge. As to the subsequent modification and qualification they undergo in the remaining retinal layers we are utterly ignorant, but there can be no doubt, from the anatomy of the structure and the rela-

<sup>1</sup> The unit  $\mu$  equals one-millionth of a millimetre.

tion of the optic tracts to the central nervous system, that the obscure changes which take place in the intervening retinal layers are of great importance to vision, with its subsidiary perceptions of light, colour, and form.

It was formerly supposed that the receptive rods and cones mechanically vibrated in unison with the subtle ethereal undulations, but such rapidity of vibration of organic formed elements is inconceivable, and this direct transference of energy without change of form or quality would appear to be physically impossible. Apart from this consideration, it is (as the Committee of the Royal Society on Colour Vision remarks) "difficult to conceive that matter which is so comparatively gross as the rods and cones which are situated on the retina, can be affected by the merely mechanical action of the vibrations of light." It is obvious, therefore, that some subtle transformation of energy between ethereal vibrations and visual impulses must take place, but the nature of this change is largely a matter of conjecture. In the course of this transformation, the occurrence of certain physical phenomena has been definitely established. Holmgren first recognised the existence of electrical processes, then Boll observed the bleaching of the retinal colour under the influence of light, which Kuehne subsequently demonstrated was the outcome of a chemical change. Again, mechanical alterations as to the distribution of the pigment in the retinal epithelium, and in the shape of the visual rods and cones, have also been observed. These varied forms of transformation of energy are evidently intimately associated with each other in the complex visual act, which involves the transformation of ethereal vibration into the special nervous visual impulse.

Nervous impulses in general are now considered to be the outcome of molecular change, generated (through the medium of some obscure chemical process) as the result of some form of physical stimulus, and propagated along a nerve-fibre. In the special nervous impulse under consideration, the physical stimulus is peculiarly subtle, and the direct transference of ethereal into molecular vibrations is not feasible on physical grounds. Whatever the mode of transformation of light energy into visual impulse may be, it is, as yet, beyond the range of tangible demonstration. Here, however, the "chemical" theory affords a particularly applicable working hypothesis, adherence to which is certainly solicited by the knowledge of the ready decomposition of many chemical substances in the presence of light. This photochemical hypothesis assumes the existence of complex unstable and therefore easily decomposable visual matters in or around the terminal visual cells, which (though not necessarily pigmented) can absorb light, but which do not generate visual impulses until decomposed under

its influence. When such decomposition occurs, chemical products are formed which are believed to excite molecular changes in the retinal elements, which changes are transmitted hence up the nerve as the visual impulses of light, colour, and form. In order that the incident light may expend its energy in provoking the necessary intermediary chemical change, it is essential that the photogenic waves be first arrested and absorbed. The minute structure of the retina demonstrates the existence of a specially differentiated brown pigment—fuscin—in the retinal epithelium, which may fulfil this function. The chief pigment of the visual cells—the visual purple or rhodopsin—was, when first discovered in 1876, regarded as the special visual matter, the subject of photochemical decomposition. Under the influence of solar light this is first changed to visual yellow (xanthopsin), and subsequently to visual white (leukopsin), while mono-chromatic spectral colours also produce the photo-chemical effect less rapidly in proportion to their absorption by the purple. It was naturally believed that the physical basis of visual impulse and colour-sensation had been isolated, and the fact that the visual efficiency of the rays corresponding to colour is relative to their photo-chemical effect upon the purple, incidentally lent support to this belief. The idea, however, had to be abandoned, for visual purple is only found in the rods, is quite absent in the cones of the human fovea centralis (where vision is most acute), and may be naturally or artificially absent in animals which see well. The visual matters are as yet, therefore, of purely hypothetical existence. Analogy suggests that some such sensitive matters (which are probably colourless and have therefore, so far, escaped detection) do exist as the physical basis of visual impulse. These, by varying photo-chemical decomposition under the influence of light of certain wave-lengths, generate corresponding impulses in the retina, which in accordance with their particular characteristics are appreciated by the perceptive visual centres as the relative colour-sensations.

The assumption that the retina is the portion of the visual apparatus mainly concerned in the differentiation of normal colour-perception is rendered very probable, and apparently justified by considerations which may be thus enumerated—

(a) *Law of Specific Energy of Nerves*—Physiological research supports the view that all nerve-impulses (actually in process of transmission) are of identical character, whatever the nature (motor, sensory, or special sensory) of the nerve may be, that the effect produced by the impulse in any given nerve depends, not upon any variation in its structure, but upon its differentiation to a special function, implying a particular peripheral termination on the one hand, and



association with a special area of the central nervous system on the other, that the nerve-fibres themselves act as indifferent conductors to the impulses which it is their normal function to transmit, that impulses in any given nerve only generate its own particular effect, whether the stimulus applied be its normal physiological one, or any other (chemical, mechanical, or electrical) capable of exciting it. Thus, a blow on the eye or section of the optic nerve produces a sensation of light, in other words, these crude mechanical stimuli can only be appreciated by the brain as the special sensation which it is in the habit of receiving through this channel.

This phenomenon strongly suggests the probability that the chemical process which initiates the molecular change in nervous impulses is identical in all nerves whatever the nature of the exciting stimulus may be, while the logical outcome of this assumption is the non tenability of those theories which, by supposing the nerve terminal to undergo different changes according to the colour striking it, endow the nerve-fibre with the faculty for transmission of a variety of nerve-impulse, and make the brain the differentiating organ.

Donders asserts that modern physiology will not countenance such a view, while the researches of Goldscheider in the demonstration of special dermal nerve-endings for the sensory impressions of touch, pain, heat, and cold strengthen, by analogy, the assumption that the retina is the differentiator concerned in colour-perception.

In furtherance of the above physiological law, Dr J Wallace has tried to demonstrate a definite relation between the length of a cone and the colour to which it responds. This implies an invariable length for each cone, which Stort has proved is not borne out by actual fact.

(b) *Uniaxial Colour-blindness*—Well-authenticated cases of this peculiar congenital condition are known to exist. Its limitation to one eye lends support to the view that the eye rather than the brain is at fault, indeed such cases can only be explained on the assumption of a defect on the peripheral side of the optic chiasma, and probably (as vision is usually of normal acuity) in the retina itself.

Professor Rutherford aptly commented to the effect that were congenital colour-defect of cerebral origin, defective colour-sense on one side of the brain would have implicated half of each eye rather than the whole of one.

The physical and physiological processes involved in colour-perception may be summarised as comprised in three factors, which are essential to the process—

1 An *external object* with its selective absorptive capacity for colour.

2 The *physical medium*—the ether—transmitting the photogenic vibrations—the specific physiological nerve stimulus.

3 The *receptive subject* with its special sensory

apparatus, provided with its specific and highly specialised end-organ. This nervous apparatus is of threefold constitution, comprising—

(a) The *retina* with its chromo-sensitive end-organs, and

(b) The *conducting nerve-fibres* for the reception and transmission of the special physical homologous stimulus to

(c) The *cerebral centres*, the special perceptive visual organ.

**THEORIES OF COLOUR-PERCEPTION**—It is not necessary to assume the existence in us of as many primary colour-sensations as there are colours in the spectrum, for it is found that in order to produce experimentally the multitudinous variety of colour therein seen, only three primary colours are essential. It is therefore generally assumed that our vision is *trichromatic*, i.e. founded on three, or the equivalent of three, primary colour-sensations. On this assumption the two main theories of colour-perception are founded.

*Young-Helmholtz Theory*—This hypothesis, propounded by Thomas Young (1807), and subsequently elaborated by Helmholtz (1852), affords a clear and simple explanation of most of the complex physiological phenomena of colour, and it is now universally accepted among physicists. It assumes the existence of three kinds of nerve-fibres identical as to structure and conduction, but terminating in different end-organs endowed with photo-chemical substances respectively sensitive to the three primary colours—red, green, and blue. In every colour-sensation these three primary colour nerve elements are stimulated, though in varying degrees, the resulting colour-sensation being determined by the proportionate amount of the three elementary sensations. In the accompanying coloured plate the relative degree of stimulation required for each of the spectral colours is indicated by the height of the colour curves and by the depth of the colour.

These three symptoms of nerve-fibres are connected with three functionally different systems of cerebral ganglion cells, whose specific energy fits them for the perception of the associated colour-sensation. The retina is thus made the selective or differentiating organ, and the brain the perceptive one.

The minute anatomy of the retina is looked upon as supporting this theory, for the longitudinal striation in the outer segments of the cones is regarded as constituting them multiple terminal end-organs. Further—the cones being the end-organs concerned in the perception of colour (Max Schultze)—the acuteness of colour-perception should be proportionate to their numerical distribution. This presumption is borne out by actual fact, for colour-perception is most acute at the macula which contains only cones, and diminishes relative to distance from

this point, while at the periphery of the retina it is quite absent

Many objections to this theory have been raised. It assumes the existence of a separate nerve-fibre to each terminal element. Salzer found about three million cones in the human retina, while there are only about one million in the optic nerve. Although as to the fovea the assumption is an essential, it is not necessary in the retinal periphery, indeed, Helmholtz explained the imperfections of peripheral vision on the assumption that several nerve elements in this situation have one nerve-fibre in common.

A further objection—originally raised by Fick and recently revived—is founded on the fact that a small pencil of rays from a distant star appears white, implying, according to the theory, that the image falls on three cones simultaneously, whereas astronomical considerations as to distance show that the tiny image cannot cover more than one cone.

This difficulty is ingeniously explained by Thomas and Welland, who suggest that the imperceptibly minute ocular oscillations constantly taking place in accordance with the rhythmical innervation of the ocular muscles, rapidly but successively expose several cones to the incident rays, and that these rapidly succeeding impressions on different colour cells are collectively appreciated by the brain as white light.

The assertion of Chaptier that this theory does not explain the fact of the central retina being less sensitive to colour than to white light is fallacious, for it assumes that while monochromatic light only stimulates one brain-cell, white light excites three equally, so that the sensitiveness of the retina to compound light is proportionately increased.

Other objections against this theory have been urged, but for the present purpose they do not require discussion.

*Hering's Theory*—This rival theory—a modification of the Young-Helmholtz—was first propounded about twenty years ago by Hering of Prague. It assumes the existence in the retina of three visual substances—white-black, red-green, and yellow-blue (in each pair one colour being complementary and also antagonistic to the other), corresponding to the supposition that we possess six fundamental sensations. As in the metabolism of living substances generally, the existence of two phases can be recognised—a constructive, assimilative, or anabolic phase, and a destructive, dissimilative, or katabolic phase,—so similar metabolic changes of opposite character in the hypothetical visual substances under the influence of light are presumed to be the causal factors in the determination of nervous impulses, and their psychological expression as conscious visual colour-sensations.

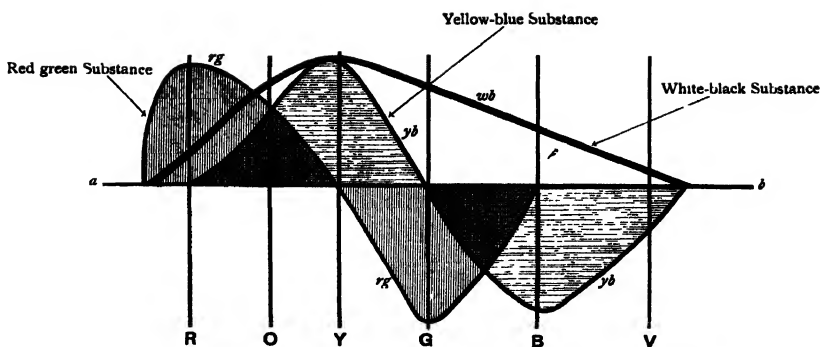
Different rays of light, according to their wave-

length, affect the several visual substances differently, provoking constructive or destructive changes in some, while others may be left in equilibrium, and producing a resulting colour-sensation expressive of the balance of effect on the whole. The accompanying diagram from Foster's *Text-Book of Physiology* (sixth edition, part iv p 95) serves to indicate the nature of Hering's assumption with regard to colour-perception.

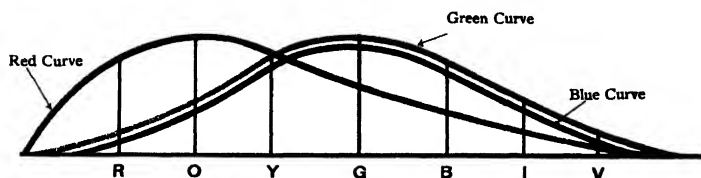
The vertical lines R, O, Y, G, B, V indicate the position on the spectrum of the corresponding colour whose initials they bear. The curved line *ag* (enclosing a space shaded vertically) represents the red-green substance, the effect of rays of different wave-length upon it being indicated by the height of the curves. The line *yb* (enclosing a space shaded horizontally) represents the yellow-blue substance, the effect of various rays upon it being indicated in the same manner. The horizontal line *ab* separates the katabolic, dissimilative, or destructive changes (represented above the line) from the anabolic, assimilative, or constructive changes (shown below it). In the red-green substance, as far as the line Y (*r* for red, orange, yellow) the effect is katabolic, producing a red sensation, beyond this point (*r* for green and blue) the effect is anabolic, producing a green sensation. In the yellow-blue substance the effect is katabolic up to G (*e* for orange, yellow, and green), producing the sensation of yellow, but beyond this point (*r* for blue and violet) it is anabolic, producing the sensation of blue. The thick line *wb* (enclosing an unshaded space) indicates the white-black substance which is only affected katabolically, though to a variable degree, by all the spectral colours, producing the sensation of white. Its anabolism produces the sensation of darkness.

Red light (R) produces the sensation of red by inducing a katabolic change in the red-green substance. Orange induces katabolic changes in both the red-green and yellow-blue substances. Yellow produces katabolism of the yellow-blue substance, the red-green now being in equilibrium (its curve intersecting the line *ab*). Green induces anabolic changes in the red-green substance, the yellow-blue now being in equilibrium. Blue produces anabolic changes in the yellow-blue substance, violet also does the same, but to a lesser degree. The simultaneous effect of mixed lights is the sum-total of the effect of their several component colours.

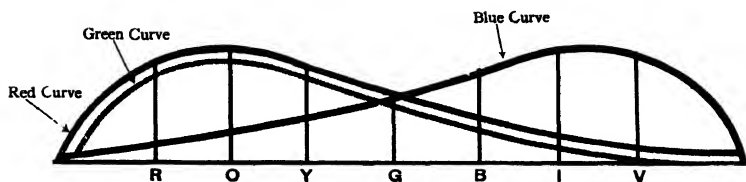
Ebbinghaus has modified Hering's theory by admitting only katabolic changes in the three visual substances as the cause of colour-sensation. He attributes colour properties to the visual purple, which latter he considers identical with the yellow-blue substance. He asserts that it exists in the cones as well as in the rods, and that its apparent absence in the former is due to its being colourless from admixture with



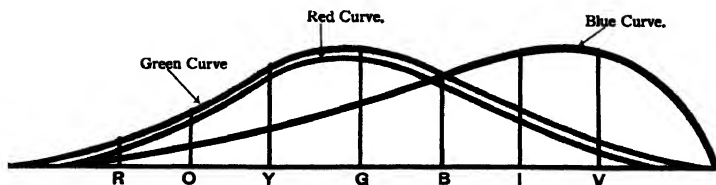
**Fig 1. DIAGRAM ILLUSTRATING HERING'S THEORY OF COLOUR PERCEPTION**



**Fig. 2. BLUE-BLINDNESS ACCORDING TO THE YOUNG HELMHOLTZ THEORY.**



**Fig. 3. GREEN-BLINDNESS ACCORDING TO THE YOUNG HELMHOLTZ THEORY.**



**Fig 4. RED-BLINDNESS ACCORDING TO THE YOUNG-HELMHOLTZ THEORY.**



the complementary red-green substance, which is present only in the cones. The outcome of this assumption, viz that the nerve of any cone transmits impulses varying according to the colour, is, however, opposed to the views of present-day nerve physiology.

The above two theories are the only hypotheses relating to colour-perception which now hold the field, and though there are still adherents to Hering's theory, that of Young-Helmholtz meets with the more universal acceptance. All other theories which endow the optic nerve-fibres with the faculty of transmitting nerve-impulses of varying characteristics are antagonistic to the accepted facts of present-day nerve physiology.

*Development of Colour-perception*—Colour-sense and colour-knowledge must not be confounded, the former is an inherited instinct, the latter results from education and practice of the sense.

Ample evidence exists that the colour-sense was coexistent with primitive man of whom we possess any trace. Among the deposits found in the sepulchral barrows of prehistoric times are variously coloured glass or quartz beads and elaborately artistic implements and pottery, while the enamelled bricks of Nineveh and Babylon and the Egyptian mummy-cases show that the ancient inhabitants of the land of the Pharaohs possessed a colour-sense of the highest order.

It is evident to any careful observer that a keen appreciation of colour is one of the marked endowments of early infancy. The colour vocabulary is absent, but education in the nomenclature of colour is alone required to voice the existence of the sense. The ordinary vocabulary for colour is a simple one—black, white, red, blue, green, yellow, grey, brown, and though the majority of educated individuals are content with so meagre a list, there is no reason why such should not be indefinitely extended. Though we have no means of knowing how certain terms were originally used or applied to designate the primary colours, the abstract nature of colour in the development of the colour vocabulary is interesting. Most of our colour-names take their origin from some concrete object. Thus the baking of a certain kind of clay gives us "brick-red," while another variety of clay gives us "terra-cotta." In "rose-colour," "cardinal," "claret," the concrete origin of the colour-name is still apparent. "Rose," "roseate," "pink," "carnation," "cherry," and "plum" are further examples, while stones such as amethyst, amber, sapphire, turquoise, topaz, and jet give us still more conclusive evidence. Probably the only difference between concrete and abstract colour-names is one of time. Orange, lilac, and even so recent an addition as that of electric-blue, are fast losing association with the objects from which the names were originally derived.

The natural colours with which civilised man is brought into contact are comparatively few, the artificially produced hues are many and increasing. A pattern-card issued by a Lyons silk manufacturer contains samples of two thousand different colours, each with its more or less appropriate name, and while this colour vocabulary is larger than the entire vocabulary of the majority of people, the gradations of colour in the solar spectrum are still more numerous. The sense to perceive these is already existent, as opportunity offers, an extended colour vocabulary will result.

*Definition*—Colour-blindness is the condition in which there is inability to distinguish certain colours. It may be *congenital* or *acquired*. The subject may be discussed in terms either of the Young-Helmholtz or Hering's theory.

*CONGENITAL COLOUR-BLINDNESS—Historical Considerations*—This congenital defect has probably been coexistent with the human race. It was so named by Brewster. The first undoubted case appears to be one recorded by Huddart in the *Philosophical Transactions* of 1777, which was subsequently commented on in 1779 by the Abbé Rozier, who also referred to the case of the painter 'Alarcen. The same year the case of Scott was reported. Then followed the historic case of Dalton, the English chemist of atomic theory fame, who in 1794 studied his red-blindness by the spectrum, and accurately described the condition. The subject was first systematically and theoretically studied by Seebeck about 1836. George Wilson of Edinburgh (1818-1859), by his researches and writings, strongly advocated attention to the practical side of the question, but his work did not receive the attention it deserved. The measure of attention which the subject in its practical bearings is now receiving is largely due to the powerful advocacy of Frithiof Holmgren, of Upsala, in his work published in 1877, and of Dr B Joy Jeffries of Boston, who wrote two years later. Yet even now, in spite of constant and consistent agitation, much remains to be done, for Governments do not yet fully recognise their duty in a matter of such vital importance to life and property.

*Congenital colour-blindness may be total or partial.*

*Total or Complete Colour-blindness—Achromatopsia*—This condition is rare, it may be unilateral (Otto Becker and von Hippel). The spectrum appears in shades of grey throughout, being lightest in the position of the yellow-green, and darkest at each end. A coloured picture appears like a photograph or an engraving. According to the Young-Helmholtz theory, such cases are explicable on the assumption that all the three photo-chemical substances are alike, as is normally the case at the extreme retinal periphery, or that the three colour-receptive elements are paralysed. Hering's

theory asserts that all the chromatic substances are absent, the white-black alone remaining

**Mono-chromatopsia**—This is a very rare group akin to the above, in which the whole spectrum appears in shades of some one colour—green or blue-violet. In such cases we assume the existence of the corresponding photo-chemical substance only

**Partial Colour-blindness**—Of this condition there are three varieties designated (according to the Young-Helmholtz theory)—blue-blindness, green-blindness, and red-blindness. All are characterised by the spectrum appearing dichromatic, with a neutral area of white or grey, as shown in the accompanying diagram

**Blue-blindness**, violet-blindness, yellow-blue blindness of Hering, akyanopsia, ananthopsia—This group is very rare, and therefore not of practical importance. The spectrum consists only of red and green, with a neutral grey area in the yellow, the blue-violet end is usually much shortened. In typical cases only red and green are seen perfectly (Erythrochloriopsia of Mauthner), blue is confounded with green, purple with red, orange with yellow, and violet with yellow-green or grey. According to Young-Helmholtz, the blue-sensitive substance is here equal to, or has the properties of the green-sensitive substance, as diagrammatically represented by the parallelism and proximity of the green and blue curves in the figure appended. From this it is evident that at Y (the point of intersection of the curves), where in normal eyes the sensation of yellow would be perceived, all the three substances would be equally stimulated, and the sensation of grey in consequence provoked

Hering's hypothesis explains the condition by the absence of the yellow-blue substance. Unocular cases have been recorded by Holmgren and other observers

**Green-blindness**, Hering's red-green blindness with shortened spectrum, achloropsia, aglaucopsia, xanthokyropsia of Mauthner—This is a group of great practical importance. The unshortened spectrum contains no green, but appears to consist of two colours only, usually called various shades of yellow and blue, which colours join each other directly, or are separated by a narrow neutral grey band. In such cases bright-green is confounded with dark-red, and a dark-green letter on a black ground is not recognised

According to Young-Helmholtz the green-sensitive retinal substance is here equal to or has the properties of the red-sensitive matter, as indicated in the accompanying diagram by the close proximity of the green to the red curve. From this it is obvious that at G (the point of intersection of the curves), where normally green would be perceived, all the three sensitive substances are stimulated equally, and the sensation of grey is brought about.

**Red-blindness**, Hering's red-green blindness with shortened spectrum, auerythropsia, Daltonism.—The spectrum is shortened owing to the absence of the red end, and is composed (as in green-blindness) of two colours—yellow and blue. The differences are that the neutral grey zone between the yellow and blue is now nearer the blue end, and that the yellow begins in the orange and not in the red as in green-blindness. In such cases light-red is confounded with dark-green, and a dark-red letter on a black ground is not recognised

The Young-Helmholtz theory asserts that the red-sensitive substance is here equal to, or has the properties of the green-sensitive substance. This is indicated in the accompanying diagram by the closely adjoining position of the red to the green curve. Here red at R is stimulated so slightly that the sensation is barely provoked, while at the site of intersection of the curves, GB, the normal greenish-blue is perceived as grey, for here all the three sensitive substances are stimulated equally

According to Hering's theory, green and red blindness are varieties of red-green blindness due to absence of the red-green visual substance, the difference in response to tests being due to variation in colour of the ocular media—the crystalline lens and the macula lutea. This is hardly feasible, for lens discoloration only occurs in senile or diseased eyes, whilst the macular pigment, which undoubtedly does affect greenish-blue rays, barely influences those concerned in the above difference

**Incomplete Colour-blindness, Diminished Colour-sense, Dyschromatopsia**—In these cases, which are of frequent occurrence, the acuteness of colour-perception is reduced. When objects are sufficiently large and well illuminated, colours are recognised, but when seen under unfavourable conditions as to illumination, or mixed with white or grey, the ground colours are less readily detected than by the normal eye. All the spectral colours are appreciated, except that violet cannot be distinguished from blue, or is called grey or brown. Pure colours are easily recognised, but when mixed, difficulty is experienced in picking out the dominant one, and gradations in colour are less obvious

The acuteness of colour-perception in persons with normal trichromatic vision is not invariably the same, indeed a difference may be noticed in the eyes of the same individual

These slight aberrations from the normal condition are probably attributable to a reduced sensibility or an alteration in the relation of the three photo-chemical substances, whereby they simulate each other in properties, and manifest their distinctive effects less potently

**Colour-ignorance**—This is ignorance as to colour nomenclature, and not inability to discriminate between colours. Its existence in the adult is very doubtful, as evidenced by the

fact that in spite of the institution of a special colour-ignorance test in the British Mercantile Marine Regulations of 1894, no failure to pass this test has hitherto been reported. It only occurs in boys of the lowest social strata whose environment has led to a total neglect of education in the naming of colours.

*Influence of Heredity and Sex on Congenital Colour-blindness*—These are important factors in the genesis of this condition. It occurs much more frequently in the male sex, and is usually transmitted from grandfather to grandson, whilst the intermediate generation—both male and female members—frequently escape the defect altogether. Several brothers may be afflicted with the abnormality, in such cases the defect usually assuming the same type in all. Very rarely transference of the defect to the female members of a family is seen. The visual acuity is usually unimpaired. Unocular cases occur, but exclusively among men.

*Prevalence of Congenital Colour-blindness*—Well-authenticated statistics of numerous observers who have collectively examined nearly one hundred thousand persons demonstrate two facts—

- (a) Its prevalence among the male sex
- (b) Its equal distribution in different nationalities.

These investigations show an average of 3.59 per cent colour-blind among males, and only .88 per cent among females. This rare occurrence in the female sex has been attributed to their special earlier individual education in colour, while this does undoubtedly account for the absence among them of colour-ignorance, it is more probable that their highly developed colour-vision is an hereditary endowment peculiar to their sex,—the outcome of a special educational development which has been in progress for ages.

*Acquired Colour-blindness*—This is a condition of defective colour-perception due to pathological changes, affecting those whose colour vision was formerly unimpaired. In the normal trichromatic eye, the visual colour-fields are physiologically limited to certain definite areas, which vary in extent according to the particular colour, and are none of them co-extensive with the field for form. Colour-perception is perfect only over a limited eccentric area surrounding the macula. Beyond this limit, deviations normally occur similar to those observed in the colour-blind throughout the whole retina—the differentiation of green, red, and blue being respectively lost, as the retinal periphery is approached, while at the extreme periphery is an achromatic zone capable only of form vision.

The distinctive symptomatic difference between congenital and acquired colour-blindness is the associated failure of visual acuity for form almost always present in the acquired

variety. The defect usually annuls or abolishes the colour-perception in a definite sequence—the order of colour-disturbance being green, red, and lastly blue. It is more frequently dependent upon pathological changes affecting the conducting nerve-fibres or the colour-sensitive cerebral cells, than to a lesion of the third factor in colour-perception, viz the retinal chromo-sensitive visual cells. This etiological fact is probably explained by the apparently greater resistance to pathological processes offered by the photo-chemical substances than by the optic nerve-fibrils, as evidenced by the observation of Stilling, who found the colour-sense unimpaired in various forms of retinitis and choroido-retinitis, and by the statement of Leber that in such retinal affections colour-blindness only ensues when the disease has led to secondary atrophy of the nerve-fibre layer and of the optic nerve.

The affection assumes a variety of types as to distribution, according to the pathological factor upon which it depends. The main varieties and the associated causes may be thus classified—

- 1 The colour defect may involve to a variable extent the retinal circumference only, and remain localised to these peripheral parts. Form vision is correspondingly restricted, the condition known as "telescopic vision" resulting. This condition follows the administration of various therapeutic agents, such as quinine, salicylate of soda, ergot, and caffeine. These drugs, from idiosyncrasy or overdose, produce toxic effects, by disturbing the vaso-motor centres and inducing a condition of retinal anæmia.

- 2 It may primarily involve the retinal periphery, and progressively spread over the whole retina. This is the most frequent form, occurring concurrently with optic nerve atrophy, whether *primary* and associated with general nervous disease, or *secondary* and consecutive to neuritis from any cause.

- 3 The defect may be limited to the central portion of the retina only, producing a central colour scotoma, which may be pericentric or paracentric according as it includes the fixation point or not. This variety includes the so-called toxic amblyopias which in reality depend pathologically upon retro-bulbar axial neuritis. The toxic agents producing this condition are alcohol, tobacco, disulphide of carbon, iodoforn, nitrobenzol, cannabis indica, arsenic, lead, stramonium and chloral hydrate. Central colour scotoma are also of frequent occurrence in disseminated sclerosis, and sometimes as the outcome of toxæmia in diabetes.

- 4 It may involve the corresponding halves of each retina (chromatic hemianopsia) as in some rare cases of cerebral hæmorrhage or embolism—the field for form being unaffected. In ordinary hemiopia the loss of colour corresponds to loss of form.

5. Disturbance of normal colour-perception is of frequent occurrence in certain functional neuroses, as hysteria, epilepsy, and hypnotism. For a detailed account of the special colour phenomena in these and in the above-mentioned groups the reader is referred to the special articles.

**Signal Colours**—By universal custom, the colours—red, green, and white—have been adopted as signals to indicate conditions of danger and safety. When the prevalence of colour-blindness in the male sex was duly recognised, it was found that most colour-blind men were either red-blind or green-blind, and the advisability of replacing the above colours by others less readily mistaken was considered. Experiments, however, as to the luminosity of various coloured lights have satisfactorily proved that the colours in usage are the best and the only available signal lights for practical purposes, for, while red transmits 10 per cent, and green 10 to 20 per cent of the light behind it, blue only allows 4 per cent to pass through, while yellow under certain atmospheric conditions would not be sufficiently distinctive. The elimination of the colour-blind from positions of vital responsibility became, therefore, a grave necessity, and upwards of forty methods for the detection have been devised.

**METHODS OF TESTING**—Any method to be scientific and conclusive must be based on the principle of the *matching* of colours, since this is the only true appeal to the colour-sense. The *naming* of colours is fallacious, for the truly colour-blind may guess correctly, while from colour-ignorance or nervousness the colour-perfect may fail. In methods involving the matching of colours, coloured pigments seen by reflected or transmitted light or the spectral colours may be employed. Of these the simplest and most practical test is that of Holmgren.

**A Wool Test of Holmgren**—Seebeck was the first to mention coloured worsteds, while Wilson of Edinburgh was the first to employ them. Holmgren's method is an elaboration of the suggestion, five finely graduated shades of each of the following colours—red, orange, yellow, greenish-yellow, green, greenish-blue, blue, violet, purple, rose, brown and grey—being employed.

The test consists in requiring the examinee to match from the heap of wools of various colours and shades (confusion colours) placed on a white cloth in a good light "test skeins" of certain definite colours. These test skeins are three in number, and are applied in a given order.

**Test 1**—The examinee is given a skein of very pure pale green (which should be held apart) and told to select from the heap all the skeins which contain any tint of that colour, care being taken to avoid specifying the colour of the test skein. If any but green skeins be selected by the examinee as a match, colour-blindness is established, while a marked dis-

position to select such, though finally not yielded to, indicates a feeble colour-sense. Should the examinee have a difficulty in understanding what is required of him, the examiner himself may show him by selecting the required skeins without in any way invalidating the value of the test.

To ascertain the kind and degree of the colour-defect two further tests may be employed—

**Test 2**—A rose-coloured skein (a mixture principally of red and blue in the proportion of two to one) is now given to the examinee, and he is asked to match it. If he select blue and purple skeins he is completely red-blind, while if he select the blue-green skeins he is completely green-blind.

**Test 3**—A bright-red skein is now presented. The complete red-blind selects as matches to this, dark green and reds, and browns (*i.e.* shades which to the normal eye appear darker), while the complete green-blind selects light green and light browns (*i.e.* shades which to the normal sense appear lighter than the test colour).

It does not always follow that the mistakes of the colour-blind are invariably those indicated above. As normal colour-perception varies in different individuals, so in the colour-blind every gradation as to kind and degree of defect exists. While typical cases of either form respond, therefore, in a distinctive manner to the tests employed, incomplete cases will be indicated by deviation from these regular lines.

Various modifications of this method have been devised for facilitating the examination and recording the results, when dealing with large bodies of men. These may be briefly enumerated as follow—1 Thomson's colour stick, 2 Jefferson's colour disc, 3 C. A. Oliver's wool test, 4 Dace's wool squares, 5 Reuss' colour table, 6 Colvin's embroidery patterns, 7 Bodal's coloured cylinders, 8 Schenke's yarn spools, 9 Donders' wool rolls.

**B Pigment Tests**—Cohn and Manthner advocate the use of bottles filled with different pigments in place of worsted.

**C The chromo-lithographic tables of Stilling** consist of coloured letters on differently coloured backgrounds, seen under reflected light. Grossmann's modification embodies this idea, but the coloured shades are shown by transmitted light.

The above-mentioned methods only permit of a qualitative determination of the colour-sense. To determine this quantitatively recourse must be had to one of several methods devised by Donders, Oliver, and Brudenell Carter.

For a truly scientific estimation of the colour-sense the spectral colours must be employed. Lord Rayleigh, Abney, Chibret, and Hirschberg have devised instruments by means of which definite spectral colours can be exposed and matched for selection from another spectrum.



**PRACTICAL REMARKS—Seamen**—The practical importance of the subject of colour-blindness was not recognised until 1855, when Dr George Wilson of Edinburgh published his *Researches on Colour-Blindness*, and graphically drew attention to the dangers of employing colour-blind men in positions of trust. In 1876 a railway disaster at Arlsey Junction, and the suggestion that it was due to colour-blindness on the part of the engine-driver, excited public attention. In the following year the Board of Trade established a compulsory test for those of the Mercantile Marine applying for certificates of master and mate. The test consisted in the naming of colours, the consequence being that men rejected at one time or place passed the test successfully on another occasion. The perfunctory manner in which the Regulations respecting colour-blind men were drawn is shown by the following facts—

1 That no test was required of "look-outs," ordinary seamen, apprentices, or pilots.

2 That the colour-blind officer received his certificate, though it was endorsed "colour-blind."

3 That no provision was made for colour-blind officers who might conscientiously feel their unfitness for the sea life.

The inefficiency of the so-called "colour-blind test" led to a long controversy between members of the medical profession and the Board of Trade, resulting finally in the establishment of the Holmgren Wool Test as the official test on September 1, 1894. The Board have not, however, yet risen to a full sense of their responsibility. The primary object of Government is to provide safeguards for human life which individual care cannot command. From this standpoint their own Reports condemn them. Between 1877 and 1894, 72,894 sailors already holding or applying for certificates were compulsorily examined; 409 failed to pass in colour. In the same period, 6370 voluntarily offered themselves for examination, and 293 failed to pass. Since 1894, when the Holmgren test came into vogue, 21,720 have been tested, with 232 failures, reduced on appeal to 215. Taking the figures of both periods together, between 1877 and 1898, 100,984 sailors have been tested, and 917 found colour-blind. Of these 917 colour-blinds there is not one tittle of evidence to show that they have abandoned the sea life. On the contrary, we know that in spite of their defect they are allowed to continue their calling, and do so to the menace of the public safety. The obvious deduction is that while the Board of Trade completely fail to procure that measure of protection which the public requirements demand, the public on their part have, by the adoption of fallacious Regulations, been deluded into a state of false security. The recommendations of the Royal Society's Committee that all candidates for positions of trust

should be compulsorily examined, and that in judicial inquiries as to collisions the witnesses should be examined for colour-blindness, would, if carried out, open an era of safety which has hitherto been unknown.

**Railway Men**—Up to the present time there has been no agreement among the various railway companies as to the application of a recognised standard colour-test. The consequence is that colour-blind men may still be found in the ranks of railway employees. Recent agitation on the part of the British Medical Association will no doubt result finally in the universal adoption of Holmgren's test, supplemented if necessary by some quantitative method.

**Colouring Matters.** See PHYSIOLOGY, TISSUES (Pigment Cells), PHYSIOLOGY, EXCRETION (Urine, Pigments), PIGMENTS OF THE BODY AND EXCRETA.

**Colp- or Colpo-**—In compound words, *colp-* or *colpo-* (from Gr κόλπος, vagina) signifies relating to or belonging to the vagina. In addition to the words specially referred to below, there are *colpalgia*, vaginal pain, *colpatresia*, vaginal atresia, *colpectasy*, vaginal dilatation, *colpeuyster*, vaginal dilating rubber bag, *colpocele*, vaginal hernia, *colpocystitis*, inflammation affecting both the bladder and the vagina, *colpo-hysterectomy*, vaginal hysterectomy, *colpo-leucorrhœa*, vaginal leucorrhœa, *colporrhoea*, vaginal prolapse, *colporrhœa*, vaginal rupture, *colpospermia*, vaginal spasm, *colpostenosis*, vaginal atresia, *colpostenosis*, vaginal constriction, and *colpovaginitis*, vaginal narrowness.

**Colpectomy.**—An extensive operation, in which the vagina is excised, and its bed columnised, the uterus may be left behind (Müller's operation) or removed as well (hystero-colpectomy); it is employed in cases of inveterate prolapse in elderly women (past the menopause).

**Colpitis.**—Inflammation of the vagina, catarrhal, follicular, emphysematous, mycotic, or other. See VAGINA, DISORDERS OF (*Vaginitis*).

**Colpocleisis.**—From Gr κόλπος, vagina, and κλείς, a key—in the operation by which the vagina is permanently obliterated or closed, it is employed in intractable cases of vesico-vaginal fistula or of prolapsed uteri (in old women). The requisites for the operation are anesthetics, vaginal specula, a uterine sound, a knife, curved needles, a needle-holder, and sutures (silk worm gut or catgut). The vaginal canal is exposed, a ring of mucous membrane is excised as high up as possible, the raw surfaces on the anterior and posterior walls thus produced are then brought together with silk worm-gut sutures, the sound being in the

bladder and the assistant's finger in the rectum during the passing of the sutures. The consent of the patient and her husband must have been obtained and the nature and result of the operation explained.

**Colpocystotomy.**—An operation by which the bladder is opened into by means of a vaginal incision, this may be done for diagnosis, for the removal of a stone or tumour in the bladder, or for intractable cases of cystitis (when the incision is to be kept open as an artificial fistula for some time).

**Colpohyperplasia Cystica.**—A degenerative change in the mucous membrane of the vagina, occurring sometimes in pregnancy, and characterised by thickening of the mucosa with the presence in it of numerous small gas-containing cysts, it was first described by von Winckel, it is probably due to a gas-producing bacillus, and is therefore really emphysematous vaginitis, and it is to be treated by glycerine tampons.

**Colporrhaphy.**—The operation (from Gr *κόλπος*, vagina, and *ράφή*, suturing) for narrowing the vaginal canal by excision of strips or areas of mucous membrane and the approximation of the raw surfaces by sutures, it may be carried out on the anterior vaginal wall (*anterior c*) or on the posterior (*posterior c*), it may be associated with perineal repair (*colpoperrineorrhaphy*), and it is commonly performed for the relief of troublesome prolapsus uteri. See PELVIS, PERINEUM AND PELVIC FLOOR (*Prolapse Uteri or Sacro-pubic Hernia*), UTERUS, DISPLACEMENT OF (*Prolapse of the Uterus, Operative Measures*).

**Colpotomy.**—The operation of opening into the peritoneal cavity through the anterior vaginal fornix (*anterior c*) or through the posterior fornix (*posterior c*), vaginal section it is a stage in various operations such as vaginal ovariectomy, vaginal hysterectomy, and vaginal hysteropexy. See UTERUS, NON-MALIGNANT TUMOURS OF (*Treatment of Fibroids*).

**Colubrine.** See SNAKE-BITES AND POISONOUS FISHES (*Colubrine Snakes*).

**Columbia, British.** See THERAPEUTICS, HEALTH RESORTS (*America*).

**Columna or Column.**—A pillar or tract or pillar-like part of the body, e.g. the columnus of the spinal cord, of the heart (*columnæ carneæ*), of the vagina, of the rectum (*columnæ Morgagni*), etc. See also BURDACH, CLARKE, GÖLL, TURCK, etc.

**Colwyn Bay.** See THERAPEUTICS, HEALTH RESORTS (*English*).

**Coma.**—A state of insensibility (from Gr

*κῶμα*, lethargy) resembling deep sleep, from which the individual either cannot be roused or can be roused only incompletely, respiration is slow and stertorous, and generally irregular. It may be due to cerebral concussion, hæmorrhage, embolism, or thrombosis, or to sunstroke, to alcoholic or narcotic poisoning, or to uræmia, diabetes, or meningitis, etc. See ALCOHOLISM (*Acute, Alcoholic Coma*), BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Hæmorrhage, Thrombosis*), BRAIN, HYPERTROPHY, BRAIN, SURGERY OF (*Concussion, Diagnosis*), DIABETES MELLITUS (*Diabetic Coma*), MALARIA (*Perniciosa Attack, Comatose Form*), MENINGITIS, EPIDEMIC CEREBRO-SPINAL, NEPHRITIS (*Clinical Features, Nervous System*), SUNSTROKE (*Heat Fever*), TOXICOLOGY (*Alcohol*), TYPHOID FEVER (*Symptoms, Nervous System*), TYPHUS FEVER (*Period of Advance, Nervous Symptoms*), UNCONSCIOUSNESS, URÆMIA.

**Coma Vigil.**—A lethargic state, in which there is unconsciousness combined with sleeplessness and (sometimes) muttering delirium (Sir W. Jenner), the phrase literally signifies "wakeful deep sleep", it is a symptom which appears generally just before the fatal termination of such diseases as typhus, delirium tremens, etc., there is no stertorous breathing, the pulse is quick, the eyes are half open, the pupils are not contracted, and there is a certain degree of consciousness. See TYPHOID FEVER (*Symptoms, Nervous System*).

**Combined Degeneration of the Spinal Cord.**—A disease of the spinal cord, occurring in cases of profound anaemia, and showing resemblances to locomotor ataxia, disseminated sclerosis, and peripheral neuritis.

**Combustion, Spontaneous.** See BURNS AND SCALDS (*Medico-Legal Aspects*).—The supposed burning of the human body from accumulation of internal heat, as in the case of drunkards, an exploded belief.

**Comedo.**—A black-head or black spot on the skin of the face due to the retention of a hardened mass of sebum in a distended sebaceous gland, the mass of sebum can be expressed by pressure on the surrounding skin, and has the form of a little yellow worm with a black head (the black head is due to dirt or to pigment). Literally the Latin word *comedo* means a glutton, and the term used to be applied to worms that devour the body. See ACNE, SKIN, BACTERIOLOGY OF THE (*The Acne Pastule*).

**Comes.**—Literally a companion, is the name given to any accompanying structure, especially a nerve or vessel, e.g. *arteria comes nervi mediani*.

**Comitialis Morbus.**—Epilepsy, so called because if a case of epilepsy occurred during the sittings of the Roman *Comitia*, there was an adjournment of the assembly, for this disease was regarded as a punishment sent by the gods

**Comma Bacillus.**—See CHOLERA, EPIDEMIC (*Bacteriology*), MICRO-ORGANISMS

**Comma Tract of Schultze.**—A tract of fibres in the posterior columns of the spinal cord in the cervical and upper dorsal regions, it lies between the columns of Goll and Burdach

**Commensalism.**—Symbiosis or the commensal state is that in which two animals, or plants, live together, the one as the tenant (but not as a parasite) of the other. It is derived from Latin *com*, together, and *mensa*, a table

**Comminution.**—The breaking of a solid body (e.g. a bone) into several small pieces (e.g. a comminuted fracture)

**Commissural Aphasia.**—Aphasia due to destruction or interruption of the connecting fibres between the different speech centres. See APHASIA (*Clinical Features, Commissural Aphasia*)

**Commis sure.**—A joining or juncture, the line where two structures meet, or a connecting band or bundle (e.g. the anterior commissure of the vulva, the commissure of the eyelids, and the posterior commissure of the spinal cord).

**Common Lodging-Houses.** See LODGING-HOUSES

**Commotio.** See CONCUSSION

**Communicated Insanity.**—The rare cases in which there is evidence that an insane person has been the cause of a similar kind of insanity in a previously sane person, folie à deux, double or induced insanity

**Compatibility.** See PRESCRIBING.

**Compensation.** See HEART, MYOCARDIUM AND ENDOCARDIUM (*Physical Signs of Different Forms of Heart Disease*), SPINE, SURGICAL AFFECTIONS OF (*Railway Spine, Compensation*)

**Complement.**—A soluble ferment which the phagocytes secrete during intracellular digestion, an alexin, a cytase, the other body which helps the anti-body to act on the micro-organism. See IMMUNITY, PHYSIOLOGY, INTERNAL SECRETIONS (*Toxic Actions and Immunity*)

**Complemental Air.** See PHYSIOLOGY, RESPIRATION (*Amount of Air Respired*), RESPIRATION (*Respiratory Rhythm and Rate*)

**Complexion.** See CHLOROSIS (*Symptoms*), SCURVY IN ADULTS (*Clinical Features*)

**Complication.**—A morbid state which arises coincidently with another disease and which is regarded as "complicating" it and modifying its course (usually in the direction of making the prognosis worse), but no hard and fast line can be drawn between true complications (accidental occurrences) and the occasional developments of a disease (e.g. nephritis in scarlet fever, dropsy in heart disease, etc.)

**Component.**—An ingredient or constituent element, e.g. in a medical prescription. See PRESCRIBING

**Compos Mentis.**—A Latin adjective phrase meaning with power over one's mind, in one's right senses, soundness of mind

**Composite Portraiture.**—A single portrait produced by combining, by superposing, those of two or more individuals, in this way an average appearance or type may be obtained, e.g. to show the typical appearance of a tubercular subject (*habitus phthisicus*)

**Composer's Disease.** See TRADES, DANGEROUS (*Lead Poisoning*), TOXICOLOGY (*Irritants, Lead*)

**Compress.**—A pad of lint or cloth so folded and arranged as to make pressure on a part or to apply water or some medicinal substance to the surface of the body, a graduated compress is one made narrower and thicker at the point where pressure is specially required

**Compressed Air Disease.** See CAISSON DISEASE, SPINE, SURGICAL AFFECTIONS OF (*Caisson Disease*)

**Compression.** See ANEURISM (*Treatment, Digital and Instrumental Compression*)

**Compression of the Brain.**—The morbid state, due to pressure on the brain of a tumour, a depressed piece of bone, or a blood clot, in which there are unconsciousness, stertorous breathing, dilatation of the pupils, and paralysis. See BRAIN, SURGERY OF (*Compression*), SPINE, SURGICAL AFFECTIONS OF (*Compression Paraplegia*), UNCONSCIOUSNESS (*Concussion and Compression*)

**Concato's Disease.**—General chronic inflammation of the serous membranes (peritoneal, pleural, pericardial, etc.) polyorrhoeitis or polyserositis

**Concealment of Birth (of Pregnancy).** See MEDICINE, FORENSIC (*Infanticide*)

**Conception.**—The act of conceiving or of becoming pregnant, also the thing conceived, viz. the ovum, embryo, and foetus. A false con-

ception is a blighted ovum, in which the gestation sac remains but the embryo has either disappeared or was never formed. See PREGNANCY, PHYSIOLOGY, etc. The words conception and false conception are also used in Psychology, with special meanings.

**Concha.**—Concha (from Gr *κόγχη*, a cockle or shell) is the name given to the shell-like part of the external ear between the tragus, the anti-tragus, and the anti-helix, it is also given to various other parts of the body, *e g* in the turbinated bones, and elsewhere. *Conchitis* is inflammation of the aural concha.

**Concination.**—A term, proposed by Duano, for tilting of the top ends of the vertical meridians of the eyes toward each other, as opposed to *divination* (tilting away from each other).

**Concomitant Strabismus.** See STRABISMUS (True, Non-paralytic or Concomitant).

**Concrescence.**—The embryological process by which it is supposed that the edges of the embryonic disc of the ovum at its posterior end are turned in, and the primitive streak carried forward and lengthened.

**Concretions.**—Deposits or calculi, consisting, as a rule, of carbonates and phosphates of lime, and forming in various organs (gall-bladder, intestine, urinary bladder, prostate, kidney, heart) and parts (joints, teeth, thrombi, etc.). See COLON, DISEASES OF (*Dilatation*), GOUT, STOMACH, INTESTINAL SAND.

**Concussion.**—The disturbance, even the complete abolition for a time, of the functions of the brain (*cerebral concussion*) or of the spinal cord (*spinal concussion*), or of both, due to violent shocks, falls, blows, etc., which apparently shake or jar the nervous tissues without causing other than microscopical lesions of the parts. See BRAIN, SURGERY OF (*Concussion*), MEDICINE, FORENSIC (*Wounds or Injuries, Fractures of Skull*), UNCONSCIOUSNESS (*Concussion*).

**Conchal Water.** See BALNEOLOGY (*Spain and Portugal*), SODIUM AND ITS SALTS (*Sulphate*).

**Condensed Milk.** See INFANT FEEDING (*Condensed Milk*), MILK (*Dietetic, Condensed*).

**Condenser.**—An apparatus for condensing the light, *e g* Abbé's or Lieberkuhn's condenser (see MICROSCOPE), or a distillate, or electricity.

**Conder's Process.**—A method for disposal of sewage by subsidence and precipitation, water enters the sewer through a ferrometer containing sulphate of iron and a slice of

lemon, the method is in use at Chichester barracks.

**Condiments.**—Accessory articles of diet which make food appetising, and so probably increase the flow of gastric juice. Some of them are aromatics (*e g* nutmeg), others are alliacious (mustard), others acid (vinegar), others salty (common salt), others sugary, and others are the peppers.

**Condom.**—A thin bag of indiarubber, caoutchouc, or goldbeater's skin worn over the male organ during coitus to prevent impregnation taking place, a check, said to be derived from the name of the inventor (Condon).

**Conduct.**—"The active or dynamic adjustment of self to circumstances" (*C. Meier, Hack Tuke*), upon deviations from the normal of conduct or behaviour the alienist has often to base his diagnosis of insanity. See INSANITY, ITS NATURE AND SYMPTOMS (*Mental Functions, Conduct*).

**Condurango.**—The bark of *Condurango blanco*, a South American vine, once recommended for cancer and still used in syphilis (not official).

**Condyle.**—Condyle (from Gr *κόνδυλος*, a knuckle) means a rounded process on a bone which serves to form part of the articulation between it and another bone, *e g* the condyles of the femur, of the lower jaw, of the occipital bone. *Condylotomy* is osteotomy of one or both condyles of the femur.

**Condyloma.** See SYPHILIS (*Secondary*), UMBILICUS, DISEASES OF (*Syphilis*), LARYNX, CHRONIC INFECTIVE DISEASES (*Syphilis*).

**Condy's Fluid.**—A red fluid said to contain 8 grains of permanganate of potash to the fluid ounce of distilled water. See DISINFECTANT, MANGANESE, POTASSIUM AND ITS SALTS (*Potash Permanganate*).

**Confectio.**—A paste containing a disagreeable medicine compounded with sugar or honey to make it less nauseous, *e g* the *Confectio Sennæ*, an electuary or conserve. See PRESCRIBING.

**Confinement.**—Detention at home or in a hospital or asylum on account of illness, it is usually regarded as synonymous either with childbirth, lying-in, delivery (*accouchement*), or with forcible detention in a lunatic asylum. See PREGNANCY, DIAGNOSIS (*Probable Date of Confinement*).

**Confluent.** See SMALLPOX (*Clinical Variations, Variola Confluent*).

**Confusion.** See GENERAL PARALYSIS (*Symptoms, Stadium Acutum*), MENINGITIS,

EPIDEMIC CEREBRO-SPINAL (*Symptoms, Psychological Disturbances*)

**Confusional Insanity.** See IN-MANITY, ITS NATURE AND SYMPTOMS (*Types, Delusional Insanity, Confusional*)

**Congelation.** See GANGRENE (*Frost-bite*)

**Congenital.**—Present at the time of birth. See AMBLYOPIA, CAPILLARIES, DISEASES OF (*Nervus*), CATARACT, CHOROIDS, DISEASES OF (*Congenital*), CORNEA (*Congenital Opacities*), DEFORMITIES (*Congenital Dislocations, etc.*), EYE-LIDS, AFFECTIONS (*Distichiasis, etc.*), HEART, CONGENITAL MALFORMATIONS OF, HERNIA (*Congenital*), KIDNEY, SURGICAL AFFECTIONS (*Hydro-nephrosis*), LACHRYMAL APPARATUS, DISEASES OF (*Blennorrhoea*), LARYNX, ACUTE AND CHRONIC INFLAMMATIONS (*Congenital Glottic Stenosis*), LARYNX, CONGENITAL LARYNGEAL STRIDOR, MEDIASTINUM (*Tumours, Congenital Cysts*), MEMORY IN HEALTH AND DISEASE (*Congenital Defects*), MENSTRUATION AND ITS DISORDERS (*Congenital Atresia*), MENTAL DEFICIENCY (*Classification, Congenital*), MORPHINOMANIA (*Congenital Habitués*), MUSCLES, DISEASES OF (*Congenital Absence*), MYASTHENIA GRAVIS (*Congenital Abnormalities*), NAILS, AFFECTIONS OF (*Congenital*), OCULAR MUSCLES, AFFECTIONS OF (*Congenital Xystasmus, etc.*), OESOPHAGUS (*Dilatation and Pouches*) PARALYSIS (*Congenital Spastic, Congenital Chorea, etc.*), PREGNANCY, INTRA-UTERINE DISEASES, RETINA AND OPTIC NERVE (*Congenital Abnormalities*), SHOULDER, DISEASES OF (*Congenital Defects*), STOMACH AND DUODENUM, DISEASES OF (*Hypertrophy of Pylorus*), TRA-TOLOGY

**Conger.** See SNAKE-BIPEES AND POISONOUS FISHES (*Fish as Hosts for Hydatids*)

**Congestion.**—Hyperæmia, or an excessive accumulation of blood in any part or organ (*e.g.* the uterus, liver, kidneys, or brain), it is not, strictly speaking, synonymous with inflammation

**Congo Sickness.** See SLEEPING SICK-NESS OR NEGRO LETHARGY

**Conhydrina.**—A vegetable alkaloid ( $C_8H_{17}NO$ ), contained in hemlock. See ALKA-LOIDS, CONIUM

**Conical Cornea.** See CORNEA (*Conical Cornea*)

**Conidia.** See MICRO-ORGANISMS (*Hypo-mycesetes*)

**Conine.** See ATRAKOIDS, CONIUM

**Conium.**—Both the leaves and the fruit of *Conium maculatum* are official. The most important constituents are—(1) *Conine*, an oily liquid alkaloid, which has a strong depressing

action on all motor nerves, and eventually on sensory nerves also, (2) *Methyl-conine*, a fluid alkaloid, with a depressing action on the spinal cord, and (3) *Conhydrine* (*qv*) The prepara-tions of Conium vary greatly as to the relative and absolute amount of these two principles present, and their effects are consequently un-reliable The preparations are—1 From *Conus Fuctus*, Tinctura Conii Dose— $\frac{1}{2}$ –1 5 2 From *Conii Folii*—(1) Succus Conii Dose—1–2 5 (2) Unguentum Conii, made from the succus (Conium has occasionally appeared to be of service in painful stomach conditions, and, as a temporary expedient, in asthma, chorea, and other spasmodic affections, when accompanied by restlessness and insomnia. It has also been used in tetanus and epilepsy, but its value in these diseases is extremely doubtful. Conine is too strongly alkaline to be given hypodermically, but a *hydroconitate* has been employed for this purpose in doses of from  $\frac{1}{4}$ –1 grain

**Conjugate or Conjugata.**—The conjugate of an ellipse is the minor axis or diame-ter, therefore at the brim of the pelvis the conjugate is the antero-posterior diameter, the anatomical conjugate is drawn from the sacral promontory to the outer edge of the upper border of the symphysis pubis, and the ob-stertrical conjugate to the inner edge, or to a point just below it, where the joint bulges slightly backward into the lum (the latter has been called the *conjugata minima*), the diagonal conjugate is measured from the sacral promon-tory to the lower border of the symphysis pubis, and the external conjugate (or Baudeloque's diameter) is measured from a point just below the spine of the last lumbar vertebra posteriorly to the anterior surface of the symphysis pubis. See GENERATION, FEMALE ORGANS OF (*Pelvis, Diameters*), LABOUR, OPERATIONS (*Induction*)

**Conjugate Deviation.**—The per-sistent turning of the eyes to one side without any alteration in their relationship to each other, *e.g.* away from the paralysed side in hemiplegia. See OCULAR MUSCLES, AFFECTIONS OF (*Paralysis*), MENINGITIS, TUBERCULOUS (*Symptoms*)

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#### ANATOMY OF THE CONJUNCTIVA

THE conjunctiva externally is continuous with the skin at the free edge of the eyelids, and internally with the Schneiderian membrane by way of the lacrimal ducts. The pinkish *palpebral conjunctiva* is firmly attached to the inner surface of the eyelids, and is connected with the ocular conjunctiva by the retro-tarsal folds, one above and one below, which by their loose arrangement prevent any dragging upon the lids by the movements of the eyeball. The *ocular conjunctiva* lies upon the anterior part of the sclera, it is pale and thin enough to allow the sclera to be seen through its texture. It is loosely attached to the underlying sclerotic, so that it may be readily picked up with forceps. By a sort of reduplication it forms towards the inner angle of the eyelids a more or less vertical fold, the *plica semilunaris*, a vestigial relic of the third eyelid of certain lower animals. The small reddish mass, often covered with fine hairs, which lies between the plica and the inner commissure of the eyelids, is called the *caruncle*. Around the cornea the ocular conjunctiva becomes closely knit to the underlying sclera, and forms what is known as the *limbus conjunctivæ*, a position in which blood-vessels are especially numerous. The conjunctiva covers the anterior surface of the cornea, but in that position it is reduced to a few layers of epithelial cells. The *nervous supply* of the conjunctiva comes from branches of the fifth cranial nerve, the *vascular supply* mainly from the posterior ciliary vessels. The *lymphatics* comprise a superficial and a deep set, with communicating branches.

#### MALFORMATIONS AND CONGENITAL ANOMALIES

*Fibro-fatty Tumour.*—*SIN Subconjunctival Lipoma or Lipo-dermoid*—This congenital anomaly is not uncommon, but as it generally causes no marked deformity, it falls comparatively seldom under the notice of the surgeon. The tumour takes the form of a soft, slightly raised yellowish mass, more or less triangular in form, and usually occupying the interval between the superior and the external rectus muscle. It lies beneath the ocular conjunctiva, and is freely movable upon the globe. As a rule, its investing conjunctiva appears normal, but short hairs may be present, a condition to which the name *trichosis bulbi* has been applied. There are a few cases recorded where a tumour occupying the customary position of a fibro-fatty growth was found to contain an osseous nucleus, in some instances covered with periosteum. I believe, as a result of microscopic investigations of these cases, that they are probably of a dermoid nature, and when they contain bone should be classed as teratoma.

*Treatment*—When inconspicuous these growths are best left alone. Otherwise, the conjunctiva may be reflected, and some of the exposed fatty-looking material sopped away with fine curved scissors. The conjunctiva is afterwards replaced, and kept in position by inserting a continuous suture.

*Dermoid Tumours*—The ordinary dermoid forms a solid pinkish-white growth, usually situated at the lower and outer part of the sclero-corneal junction, and encroaching to a variable extent upon both cornea and conjunctiva. It is attached firmly to the cornea. Hairs may or may not grow from the dermoid, the top of which is now and then dry and fatty-looking. The tumour (generally limited to a single eye) is usually about the size of a split pea. A case has been reported, nevertheless, where the growth had reached the bulk of a horse-bean, while upwards of twelve long hairs grew from its middle part, passed between the eyelids, and hung upon the cheek. The patient remarked that these hairs did not appear until he was sixteen years of age, at which time also he has beard grew. The dermoid often shows a distinct tendency to get larger, and to become hairy at or about puberty. The microscope shows that these growths include the elements of ordinary skin, as epithelium, glands, hair follicles, etc.

*Treatment*—The growth may be removed by careful dissection.

*Nævi*—Angiomata, plexiform or cavernous, may involve the conjunctiva, and are usually associated with a similar condition of the eyelids, orbit, face, or of other parts of the body. The commonest clinical appearance is that of a small or large patch of purplish-red discoloration affecting the palpebral mucous membrane.

More rarely a definite tumour is met with, involving, it may be, the semilunar fold or the ocular or the palpebral conjunctiva. In some of these cases the angioma forms a livid, nodulated mass, bleeding on slight provocation.

**Treatment**—Small capillary nevi, which give rise to no disfigurement, call for no special treatment unless they show a tendency to get bigger. More conspicuous patches may be seared with the galvano-cautery, or (after carefully drying the parts) be painted lightly with solution of sodium ethylate (B.P.) until improvement results. The cavernous angiomas are best dissected out, an operation that is by no means so difficult as it seems.

**Lymphatic virus** is a rare but interesting congenital condition which depends upon dilatation of the lymphatic vessels, with hyperplasia of the conjunctiva. Some part of the ocular mucous membrane is occupied by a bunch of small yellowish elevations, which convey the notion that they contain fluid. The mass may undergo variations in size, particularly in females at the menstrual period. In a case of my own the appearance reminded one of a non-inflammatory chemosis, of yellowish colour, occupying the ocular conjunctiva on the nasal side of the eyeball. In another case a prominent mass of yellow-like tissue, dotted over with tiny hemorrhagic points, was connected with the semilunar fold, while in the same eye there was a collection of yellowish beaded vessels in the upper part of the ocular conjunctiva. Both lids were thickened, presumably from lymphatic obstruction. The eyeball was small, shields of persistent pupillary membrane were present, dotted opacities were diffused through the crystalline lens, and sight was extremely poor. Ait examined microscopically an instance of lymphatic virus, and found in the ocular conjunctiva a series of canals and cavities, the walls of which were lined by endothelium and formed by the compressed fibres of the conjunctival tissue.

**Moles** have been described as existing upon the ocular conjunctiva.

**Supernumerary Caruncle**—It appears that the caruncula lacrymalis, like the tragus or the nipple, may be duplicated. The first observation of the kind was published by the writer in 1896, and since then Dr J. W. H. Eyre has recorded two similar cases. A small, raised, fleshy mass, of granular appearance, is attached to the conjunctiva of upper or lower lid somewhere near the normal caruncle, fine hairs may be present on the surface of the growth.

**Treatment**—Should it be desired for æsthetic reasons, the little tumour may readily be snipped away with scissors. Recurrence is unlikely.

**Other Rare Affections**—The writer has seen perhaps half-a-dozen instances of a peculiar congenital growth, which, so far as he is aware, has not been mentioned in literature. A greyish

red tumour, oblong or reniform in outline, occupies some part of the ocular conjunctiva. It is freely movable with the conjunctiva in which it lies, a few dilated vessels often run towards it. Microscopically it seems to be of cystic nature, its wall being formed of a thick but irregular stratified epithelium, together with a vascular subepithelial layer of connective tissue. The tumour is easily dissected out, and if the wound be carefully closed with a continuous suture, scarcely a trace of the operation remains.

#### INFLAMMATORY AFFECTIONS

The conjunctival sac, even in a state of health, is seldom altogether free from micro-organisms, of which by far the commonest is the so-called xerosis bacillus, the staphylococcus pyogenes albus (or the staphylococcus epidermidis albus) is also often found. Certain other microbes are present under morbid conditions, as, for example, gonococci, Weeks' bacilli, pneumococci, diplobacilli, bacillus coli communis, diphtheria bacilli, and various pyogenic cocci. They give rise to definite conjunctivitis, the diagnosis of which is nowadays made chiefly from a bacteriological examination. Although our knowledge of the parasitic nature of acute ophthalmia is fairly complete, the same cannot yet be said of the chronic forms, especially of trachoma.

#### ACUTE OPHTHALMIA

As stated above, in our present state of knowledge it is possible to ascribe nearly every known form of acute ophthalmia to the action of specific microbes. At the same time, to avoid confusion, we may retain the classical clinical divisions and speak of 1 Mucopurulent ophthalmia, 2 Purulent ophthalmia, 3 Granular ophthalmia, and 4 Membranous ophthalmia.

**1 MUCCOPURULENT OPHTHALMIA**—This common affection tends to become epidemic in spring and summer, and at times it is extremely contagious. No age is exempt, although it is, above all things, a childish ailment.

**Etiology**—It is associated with several distinct micro-organisms, of which two are more widely spread than the rest put together, namely, (a) the short and slender bacillus described by Koch and Weeks, and (b) the diplobacillus to which attention was directed by Morax a few years ago. In some other countries, however, it would appear to be more generally due to pneumococci. It is sometimes set up by the bacillus coli communis, as well as by the various pyogenic cocci, as the staphylococcus aureus, citreus, and albus, although in the case of the last-named a special predisposing condition of conjunctiva is probably essential to the ophthalmia. I have met with one case (associated with an evil-smelling discharge) where the bacillus pyogenes fetidus appeared to be the

cause. But, on the whole, we may with tolerable confidence say that a severe muco-purulent conjunctivitis in this country is likely to be due to Weeks' bacillus, whereas a much milder subacute form is probably associated with the diplobacillus of Morax. In my experience, the other organisms named above are comparatively rare as causes of conjunctivitis.

**Characteristics.**—The upper lids are reddish and puffed, the ocular conjunctiva is bloodshot and perhaps ecchymosed, the palpebral conjunctiva is uniformly red and rather swollen, and its so-called "follicles" (see page 209) are often peculiarly prominent. Yellowish discharge is found about the eyelashes, at the inner canthus, and, mixed with lacrimal fluid, in the recesses of the conjunctiva. Chemosis is seldom a marked feature. Phlyctenulae about the conjunctiva or elsewhere may be present. The patient complains of his eyes feeling hot and heavy, as well as of a sensation which he often likens to sand or dust in the eye, a degree of photophobia is not rare. In uncomplicated cases the cornea is clear, the iris bright, and the pupil mobile. Speaking generally there is little tendency to corneal mischief, especially in children. When the inflammation is caused by the pneumococcus there is apt to be a thin, fibrinous deposit upon the conjunctiva of the upper lid. The special sign of diplobacillary inflammation (in addition to its subacute character) is angular blepharitis—that is to say, an excoriation and soreness of the inner and outer canthus. Pus infections are observed in children suffering from a coincident impetigo or discharge from the ear. The bacillus coli sets up an acute conjunctivitis which has no distinguishing feature beyond its tendency to subside spontaneously.

**Treatment.**—The principles are—first, to kill the pathogenic organisms by the local application of antiseptics, secondly, to remove morbid secretions, and thirdly, to relieve special symptoms, such as pain. The best remedy for all severe cases is a 2 per cent solution of silver nitrate applied to the exposed conjunctiva once a day. The acute symptoms seldom withstand more than three or four applications, but the remedy should be persevered with until microscopical examination shows discharge to be free from the specific organisms. The same agent succeeds in most of the remaining acute muco-purulent inflammations of the conjunctiva, except possibly in that due to the diplobacillus. The latter is best treated by means of a strong solution of zinc sulphate (gr 10 to the ounce) dropped over the conjunctiva three or four times a day. Chloride of zinc (gr 5 to the ounce) may also be used with success. For removing discharge from the eye, a saturated solution of boric acid or corrosive sublimate (1 5000) succeeds admirably. The liquid should be applied at or about body temperature, and the lids, whenever possible, should be everted. Pain, when

at all severe, may be relieved by the local use of a 1 per cent solution of cocaine hydrochloride, dropped into the eyes at intervals of a few hours. In severe cases it may be necessary to give bromide of potassium or chloral internally.

**2 PURULENT OPHTHALMIA.**—This ailment results from the gonococcus reaching the conjunctival sac. It is extremely contagious. *Pathologically*, it is characterised by dense cellular infiltration of the subepithelial tissue, and by great thickening of the surface epithelium, in which, by suitable means of hardening and staining, active karymitosis can be seen. The gonococci do not penetrate the mucosa much deeper than the epithelium, most of which is sooner or later shed. The following points may be noted with regard to gonococci and their relationship to cases of purulent ophthalmia—(a) They are scanty to begin with, but become numerous when the discharge gets profuse, (b) the thicker the secretion the greater their number, (c) they are not seldom mixed with xerosis bacilli and with pus organisms, (d) they may persist for several weeks. In practice purulent ophthalmia is met with under two forms, as it affects (1) newly-born children, and (2) older subjects. The clinical differences between these varieties render a separate description desirable.

**(1) OPHTHALMIA NEONATORUM.**—This usually develops on the second or third day after birth. Earlier cases are attributable to a lingering labour in which the infant has been infected during the act of parturition, and later ones to inoculation from contaminated lochia. Material in the maternal passages, containing gonococci, clings to the baby's lashes, and is carried into the eye after birth, either by the blinking of the infant, or by the sponges, water, or other articles employed in the first bath. Its leading feature is a discharge, at first thin and whey-like, and later resembling pus. As the case advances the eyelids are greatly thickened by serous effusion, and when thus swollen they tend to become everted when the baby cries. The palpebral conjunctiva (often difficult to expose) is red, thick, folded, and has a villous look "like a finely-injected fetal stomach" (J C Saunders). Chemosis, however, is not common. In this dangerous affection the cornea may be rapidly involved, particularly in premature or syphilitic infants, an abnormal tightness of the eyelids also is unfavourable to recovery. The baby, in marked cases, is often fretful, while diarrhoea is apt to come on, especially if the infant is bottle-fed. The complications include swelling of the preauricular glands, abscesses about the eyelids, purulent discharges from the external genitals of female children, arthritis, and (rarely) inflammation of the serous membranes. Common results are cicatricial changes in the conjunctiva, opacities of the cornea with or without inclusion of the



iris, anterior capsular cataract, nystagmus, and squint.<sup>1</sup>

**Treatment** — Discharge must be removed frequently by means of antiseptic lotion, as corrosive sublimate (1 5000), mercuric cyanide (1 1000), potassium permanganate (1 1000), naphthol (1 5000), boric acid (saturated solution), or chlorine water. The application should be warm, and applied to the everted conjunctiva with a morsel of absorbent cotton-wool. A syringe should not be used for the purpose, since it is capable of doing mischief to both patient and operator. In bad cases, during the height of the attack, the baby must be tended by the nurse day and night, as cure depends chiefly upon the care with which the eyes are kept clean. Iced applications to the eyelids find favour in some quarters, but they call for much attention, tend to confuse pus, and are difficult to keep in place. On the other hand, the palpebral conjunctiva may be painted with a 2 per cent solution of silver nitrate irrespective of the stage of the disease. The solid silver stick, whether mitigated or otherwise, cannot be recommended, as its use is almost certain to be followed by ulceration of the conjunctiva. The silver solution may be applied once a day, or even twice in severe cases. There are two other remedies that may be substituted for silver nitrate, namely, protargol (50 per cent) and largin (5-10 per cent), both of which are synthetic compounds of silver and proteid substances. They are used in the same way as the silver nitrate — that is, upon lids everted and carefully freed from discharge. The use of one or other of the foregoing preparations should be continued until the discharge gets thin and scanty, and is found no longer to include gonococci. The remedy is then to be employed twice or thrice a week for a longer term, so as to avoid all risks of relapse. If the cornea be hazy when the case comes under notice, physostigmine should be dropped into the eye three or four times a day. — Physostigmin sulph., gr 2, cocaine hydrochlor., gr 5, distilled water, 1 ounce. Atropine sulphate (gr 2 to the ounce) may be used instead of physostigmine when the cornea is actually ulcerated. Should the ulcer be deep or have yellow edges, or should there be pus in the anterior chamber, then more vigorous measures must be taken without further delay. For example, an anæsthetic should be given, and the ulcer either freely scarfed with the galvanocautery or else thoroughly touched with the liquefied carboic acid (B.P.). These operations may have to be repeated.

(2) **PURULENT OPHTHALMIA IN OLDER SUBJECTS** — This affection differs from ophthalmia

<sup>1</sup> It must be borne in mind that forms of ophthalmia other than those caused by gonococci are met with in babies. The writer has found gonococci in 66 per cent of his cases, in the others, Weeks' bacilli, diplobacilli, pneumococci, or bacillus coli communis appeared to be the cause of the inflammation.

neonatorum in the following respects — (1) It is generally confined to a single eye, (2) it affects men more often than women, (3) its prognosis, especially in persons over twenty years of age, is very grave. Its most frequent cause is auto-inoculation from a coexisting gonorrhoea. The virus is spread by fingers, washing materials, bed linen, or contaminated instruments or dressings. In hot countries flies appear to be the most usual agents of infection.

**Symptoms** — After an incubation period averaging about two days, the disease declares itself by pain, photophobia, swelling of the lids, and the discharge of thin, whey-like material from the conjunctiva. The early signs are so little characteristic in the absence of a clear history of inoculation that the case may be readily mistaken for one of catarrhal inflammation, unless the secretion be examined with the microscope and found to contain gonococci. But before long the conjunctiva of the eyelids becomes markedly swollen, villous, and of a deep-red hue, that of the eyeball is infiltrated with serum, so as to form a mound of jelly-like thickening around the cornea (chemosis). Lacerating pains are common, and the eye may be extremely tender. Meanwhile the abundant discharge is thick and yellow, and includes innumerable gonococci. After a few days the swelling of the lids and conjunctiva becomes less marked, and in most cases the condition slowly recedes. The process, however, when severe, almost always sets up more or less conjunctival scarring. The cornea may suffer either early or late. The earlier and much more dangerous ulcerations are to be suspected when there is much chemosis, but are generally discovered only when the swelling of the eyelids has begun to go down. A dulness is observed either in the interpalpebral zone or else in the central region of the cornea, and, while in a few instances the process may go no farther, usually the greyish area becomes converted into an actual ulcer. The latter may be clear or have a yellowish look, in which event it is likely to perforate the cornea, a staphyloma resulting. The later ulcerations may be central or peripheral, but suitable means will generally prevent their spread, especially when they show any tendency to vascularity. The affection, like ophthalmia neonatorum, may be associated with, or followed by, inflammation of the joints, as in a case related by Weiss and Klingelhoefer. Indeed, there is no reason why the other complications named on a former page should not also occur.

**Treatment** — In principle, the treatment of ordinary gonorrhoeal ophthalmia does not differ from that of the same affection in babies. The results, unhappily, are nothing like so favourable. The patient must be put to bed, and the pain relieved by sedatives or narcotics

During the earlier stages good compresses of corrosive sublimate lotion (1:5000) may be applied to the swollen lids. Discharge must be removed with one or other of the weak antiseptic lotions mentioned. The plan of irrigating the conjunctival sac with large quantities of a diluted lotion of permanganate lotion with a special irrigator deserves a trial. Gonococci must be destroyed by the application, once or twice a day, of silver nitrate (2 per cent), protargol (50 per cent), or largin (5-10 per cent). Those remedies, however, must be used with great caution until the discharge assumes the characters of pus. Corneal complications must be treated with atropine drops (gr 2 to the ounce), or with the galvanocautery, as described elsewhere. Should a "granular" state of the conjunctiva succeed the acute disorder, the sulphate of copper stick, applied daily, will be found the best remedy. When one eye alone is affected an attempt must be made to save the other from invasion. The most satisfactory plan may be briefly described as follows—A watch-glass crystal is enclosed between two pieces of adhesive plaster, in which holes have been cut. This contrivance (introduced by Dr Buller) is then fastened in front of the sound eye, which can by those means be inspected at intervals without the risk of its being touched by fingers contaminated with gonococci. As a further safeguard, the patient, as far as may be, should lie upon his affected side.

**3 ACUTE GRANULAR OPHTHALMIA**—Granular ophthalmia or trachoma (*v* p 209) is essentially a chronic affection, subject to acute exacerbations. The clinical appearances in acute cases vary much, but one feature is common to all, namely, the development in the palpebral conjunctiva of many "sago-gran" granulations. A bacteriological investigation of the secretion has in about two-thirds of my cases revealed the existence of an organism indistinguishable from Weeks' bacillus, and this is doubtless the microbe recently described by Dr Leopold Muller as the specific cause of the malady. Its presence may be explained by an outbreak due to Weeks' bacillus occurring in a community where trachoma was prevalent. A mixed infection is thus set up, which manifests the clinical picture of an acute or subacute trachoma. The disease is invariably followed by chronic trachoma.

*Treatment* is that of muco-purulent ophthalmia, and, later, when chronic, that of trachoma. It must never be forgotten that acute trachoma is one of the most contagious inflammations of the eye, so that early isolation is of vital importance.

**4 MEMBRANOUS OPHTHALMIA**—Several distinct forms of ophthalmia may be associated with a membrane upon the conjunctiva. The Klebs-Löffler bacillus may give rise to a slight or to a severe form of inflammation under

conditions that are as yet obscure. The milder form has for years been known as *croupous*, and the more severe and less frequent as *diphtheritic ophthalmia*. The two affections are more frequent in young children, and tend to follow closely zymotic ailments, such as measles and scarlet fever.

(1) *Croupous ophthalmia* is not unlike a rather severe form of muco-purulent conjunctivitis set up by Weeks' bacillus. The lids, often a good deal swollen, can be readily everted, and strings of glutinous secretion frequently stretch from one lid to the other. The ocular conjunctiva is congested, and may be slightly thickened. The striking feature of the affection, however, lies in the grey membranous exudation upon the palpebral and (more rarely) upon the ocular conjunctiva. The membrane can be stripped away, exposing beneath a thickened, red, and bleeding surface. There may be spots of diphtheritis about the lids, nose, or face, the preauricular and angular glands may be involved, the general health is usually good. The disease is not followed by symblepharon or by cicatricial changes in the conjunctiva. The prognosis is favourable. Croupous ophthalmia is comparatively common in London.

(2) *Diphtheritic conjunctivitis* has a clinical appearance so characteristic that a diagnosis can often be made at sight. The discharge, at first thin and scanty, after a few days turns to a thinish pus, in which lie shreds of necrotic tissue. If the swollen lids can be everted, the palpebral conjunctiva will be found firm and lardaceous, or "brawny," either throughout or in patches. A marked feature is the presence of depressed, greyish-white areas, associated with small, dark-red, ecchymotic spots. The bulbar conjunctiva and the cornea may be covered with false membrane. The affection is followed by cicatricial changes and deformities of the lids. Diphtheria of the fauces or elsewhere may precede, accompany, or follow the ophthalmia. The patients, who are seriously ill, show such symptoms as raised temperature, frequent pulse, depression, anaemia, albumin in the urine, and loss of knee-jerks. A sequel is peripheral neuritis, as shown by palsy of the palate, ocular muscles, extremities, and so on. The disease is very rare in England.

*Treatment*—If the clinical evidence of diphtheria is strong, antitoxin should be used without waiting for the results of a bacteriological examination; otherwise the injection may be deferred for a time. Where Klebs-Löffler bacilli are found, antitoxin should be immediately administered. The effect of the remedy, when used early, is remarkable. Experience has proved that antitoxin alone effects a cure. It is advisable, however, to employ local treatment as well—first, to hinder the absorption of toxins, and, secondly, to destroy organisms,

other than Klebs-Löffler, that may be present. For this purpose, in the earlier stages, the writer prefers a 15 per cent solution of potassium permanganate, and, later, a 2 per cent solution of silver nitrate. The remedy selected must be applied once or twice daily, if possible, to the everted conjunctiva. Meanwhile the eyes must be kept clean with an antiseptic lotion, such as corrosive sublimate (1:5000), or boric acid (1 per cent), or quinine. A good formula is quinine hydrochloride, gr 2, distilled water, 1 ounce. Stimulants are often needed in severe cases. The patient must be carefully isolated, especially from other children.

#### CHRONIC OPHTHALMIA

Three forms of conjunctivitis are included under the general description chronic ophthalmia, but before describing them, a few words must be said with regard to the healthy palpebral conjunctiva. The lower tarsal membrane is generally traversed by arborescent vessels, and shows, especially towards its outer side, a number of minute transparent elevations, the so-called "follicles," which are neither more nor less than collections of small round cells. Another common appearance consistent with health is that of a single row of minute elevations, which lie close to, and parallel with, the free edge of the outer third of the eyelid. The upper tarsal conjunctiva may be smooth or slightly velvety, particularly along its lower convex edge. It is of so thin a texture as often to allow the underlying Meibomian glands to be recognised as so many vertical markings. It is quite common for a few tiny elevations to be at the corners of the upper tarsal conjunctiva. Deeply-seated vessels may be seen shining through the loose superior retro-tarsal folds. "Follicles" are frequently to be distinguished, and the lobules of the inferior lacrimal (or palpebral) gland can always be found embedded in the outer part of the folds. It is most important to study the characters of the normal conjunctiva attentively, for a want of that knowledge is likely to lead to errors both of diagnosis and of treatment.

**1 CHRONIC MUCO-PURULENT CONJUNCTIVITIS.**—This affection results in most cases from a neglected acute catarrh. Its subjective symptoms include heaviness and dryness of the eyes at night, frequent blinking, and sensations like those of foreign substances in the eye. Indeed, they are often like those of an uncorrected error of refraction. The palpebral conjunctiva may be unduly red, secretion may be present after sleep; and a common appearance is that of a whitish, frothy spume collected in the corners of the eye. The patients frequently complain of colours around artificial lights, the result of particles of mucus on the cornea. Complications—as, for example, troublesome lachrymation, blepharitis, and ulcerative keratitis—are more common in elderly subjects.

**Treatment.**—Wind, smoke, dust, or impure air must be avoided. Errors of refraction or muscular anomalies should be corrected with suitable glasses. The lacrimal passages and the nose must be carefully examined. Weak solutions of silver nitrate (gr  $\frac{1}{2}$ -1 to the ounce) often render yeoman service, although they must not be employed for too long, owing to the risks of staining the conjunctiva. Other useful local astringents are zinc sulphate (gr 1-2), alum (gr 2), boric acid (gr 10). In many cases it is advisable to add to the foregoing lotions a little tincture of opium. A remedy in much favour is the yellow lotion<sup>1</sup> of the Austrian Pharmacopœia, dropped into the eye twice or thrice a day. In children by far the best remedy is the familiar yellow oxide of mercury ointment (gr 10-20). An ointment of copper sulphate, containing  $\frac{1}{2}$  to 2 gr of the precipitated salt to half an ounce of soft paraffin and an equal amount of hydrous wool fat, is often efficacious. In difficult cases the everted conjunctiva may be touched occasionally with solid alum or bluestone.

**2 FOLLICULAR CONJUNCTIVITIS.**—The symptoms of this affection resemble those of chronic muco-purulent conjunctivitis, but in addition the "follicles" are unusually prominent. These are rounded or oval, seldom exceed 1.5 mm in diameter, are more or less transparent, and are generally arranged in rows "like the beads of a rosary." The conjunctiva is rarely deeply involved, and the changes are always more marked in the lower lids than elsewhere. The condition is never associated with consecutive corneal changes. Its predisposing cause appears to lie in the adenoid tendency of certain subjects, its exciting cause may be either general, as insalubrious surroundings, or local, as an attack of acute ophthalmia, the prolonged use of atropine or physostigmine, and so on. The best name for this widely-spread condition seems to be *simple folliculitis*.

**Treatment.**—If the "follicles" give rise to no symptom, and are not associated with discharge from the eye, they are best left alone. Otherwise, the same remedies may be employed as described in the last section. Tannate of lead ointment (10 per cent) often acts well. If the overgrowth be large, there can be no objection to getting rid of them by squeezing, cutting, or other surgical means. A change of air is sometimes beneficial.

**3 TRACHOMA.**—This important affection is found almost exclusively amongst the poorest classes, and in schools attended by those classes. It may cling to a particular building for years, simply because proper steps are not taken to

<sup>1</sup> The yellow lotion contains ammonium chloride, 0.5, and zinc sulphate, 1.25 parts, dissolved in 200 parts of distilled water. To that solution is added a mixture of absolute alcohol 20 parts, camphor 0.4, and saffron 0.1. The two solutions are mixed, allowed to stand for twenty-four hours, and filtered.

eradicate it. Certain races—as Russians, Poles, Armenians, Jews, and Irish—are peculiarly subject to the disease, probably because their mode of life favours the spread of infection. Negroes, curiously enough, are said to be practically exempt. In some countries, as Switzerland, trachoma is almost unknown, in others, as Belgium, it is widely spread. It arises only by infection, but we know nothing definite as regards the pathogenic agent, thought by some to be a micro-organism, and by others to be a protozoan. This much, however, is certain—that infection is fostered by a gregarious life, by bad ventilation, by lack of cleanliness, by defective washing arrangements, and by the absence of isolation for those affected. Indeed, the clinical evidence of the transmissibility of trachoma is overwhelming. Both eyes are usually affected sooner or later.

**Symptoms.**—Trachoma is essentially a chronic ailment, subject (particularly in the earlier stages) to acute or subacute exacerbations. To begin with, it affects the palpebral conjunctiva, but, later, may spread to the cornea. Its leading features are—first, the development of “sago-grain” bodies in the conjunctiva and, secondly, thickening of the palpebral mucous membrane. The “sago-grain body,” which is almost essential to diagnosis, appears to be a “follicle,” altered as the result of specific invasion, presumably by a micro-parasite. It is a round, opaque, greyish-white growth, deeply embedded in the hypertrophied conjunctiva, especially of the upper cul-de-sac. Microscopically, it consists of grouped lymphocytes, supported by a delicate stroma, and often more or less distinctly encapsulated. According to some recent researches, it may originate in the lymph-vessels or spaces of the conjunctiva. Discharge, except in acute cases, is seldom abundant. An almost constant sign of trachoma is ptosis, which gives the patient a sleepy look. The cornea may suffer in two ways, namely, by the development of pannus or of ulceration. A well-marked pannus gives a characteristic appearance to the upper third or so of the membrane. The affected parts are irregular and cloudy, and few or many enlarged vessels (continuous with those of the conjunctiva) branch out into the web. In bad cases the entire cornea may suffer, and vision be reduced to bare perception of light. Indolent ulceration is not uncommon along the free edge of a pannus. The usual explanation of the origin of pannus assumes that it is due to the irritation of a rough and thickened upper lip. This theory, nevertheless, is hardly borne out by the known facts. Without entering into contentious points, it may be safely asserted that pannus represents an actual trachoma of the cornea, brought about either by direct inoculation from the affected lid, or else by the metastasis of infective particles through vessels or lymphatics. Ulcerations of

the cornea simply mean disintegration of the diseased material. The sequels of trachoma include scarring of the palpebral conjunctiva, shortening of the cul-de-sac, trichiasis, entropion, and imperfect sight as the result of corneal blemishes.

**Treatment.**—*Escharotic, surgical, and accessory means* are used. For general use, the best *escharotic* is solid bluestone, rubbed over the exposed palpebral conjunctiva once a day. The application may be continued for years without harm. Where inflammatory symptoms are marked, 2 per cent solution of silver nitrate suits admirably, but it should be replaced by bluestone as soon as possible. Other *escharotics* are—corrosive sublimate solution (1 to 4 per cent), solid lapis divinus, and undiluted carbolic acid. *Operative measures* are to be adopted under the following circumstances—(1) When large masses of granulations are present, (2) when the cornea is affected, (3) when local treatment has been tried for several months without success, (4) when a patient is unlikely to submit to a prolonged course of escharotic treatment, (5) when one eye is alone involved. The various operations include expression, partial removal of the upper retro-tarsal folds, and scrubbing the palpebral conjunctiva with a tooth-brush dipped in some antiseptic lotion. The chief *accessory measures* are the use of such lotions as previously mentioned, and the treatment of corneal ulcerations.

#### PHLYCTENULAR CONJUNCTIVITIS

This condition is common in weakly children of the poorer classes, and frequently develops after measles or other zymotic disease. In many cases it is dependent on “scrofula” or “tubercle.” Bach believes that it is set up by pyogenic microbes, particularly the staphylococcus pyogenes aureus. He has succeeded by inoculation in producing the same affection, not only in rabbits, but also in human beings. Its chief importance is derived from the fact that it is often associated with a similar affection of the cornea. It is extremely prone to relapse, unless its cause can be made out and remedied. Knie has suggested that it should be termed “eczematous” when it coexists with eczema in other parts of the body, but that when such is not the case the neutral term “phlyctenular” should be retained.

**Symptoms.**—One or more small pimples appear upon the sclero-corneal junction, and a triangular leash of vessels, having its apex at the affected spot, is generally present. The little nodul speedily loses its epithelium, becomes converted into a superficial ulcer, and then heals completely. It may, however, resorb without a any time ulcerating. When phlyctenule lies in the ocular conjunctiva, at some distance from the limbus, they are usually large and accompanied by muco-purulent secretion. In children their

are several atypical forms of phlyctenular disease that need not be described more particularly.

**Treatment**—Improve the general health by good food, fresh air, cleanliness, outdoor exercise, plenty of sleep, and (whenever possible) by residence at the seaside or in the country. Cod-liver oil, best given as an emulsion, is of special value, and tonics, as Parrish's syrup and the syrup of ferrous iodide, are useful. The use of these remedies should generally be preceded by a short course of mercury, best given as Hydrate of mercury, gr 1, pulv rhei, gr 1, sodii bicarb, gr 2, one powder every night for a week. Locally, calomel<sup>1</sup> may be sprinkled over the injected parts once or twice a day, or the same remedy may be given for home use as an ointment containing 20 grains to the ounce. Yellow ointment (gr 10 to the ounce) suits many cases, and may be used for months together where there is a marked tendency to relapse. Should photophobia be prominent, atropine or cocaine may be combined with the mercurial preparation.

**SPRING CATARRH**—This rare form of disease is important from its liability to be confused with trachoma. Its signs and symptoms, however, are quite characteristic. It is marked by firm greyish elevations in the limbus, as well as by a pale condition of the palpebral conjunctiva, which is so closely beset by enlarged papillae as almost to recall the appearance of a cobble pavement. The palpebral conjunctiva looks as though it were coated with a thin layer of milk (Vetsch). The patient complains of great irritability of the eyes of a distinctly seasonal type. The irritation begins in spring or early summer, and, after subsiding more or less completely during the winter, returns during the following spring. Spring catarrh usually affects both eyes, although not necessarily to the same degree. It is met with mainly in young persons, and may persist for many years. Nothing definite is known as to its pathology.

**Treatment**—Lotions containing boric acid, sulphate of zinc, or antipyrine are useful. Ointments of salicylic acid or corrosive sublimate (gr 1-2 to the ounce of lanoline-vaseline), or of ammoniated mercury (2 per cent), may be employed. In some cases douches of hot water appear to give temporary relief. If photophobia exist, the eyes should be protected with goggles. Surgical measures have been recommended, but are not in favour with most ophthalmic surgeons. Arsenic may be given internally. For my own part, I have seen more benefit from change of air than from any other means, local or general.

**EXANTHEMATOUS CONJUNCTIVITIS**—By this name we mean any inflammation of the conjunctiva associated with or closely following an exanthem. Measles often gives rise to such a

condition, which is characterised by its tendency to relapse and its rebelliousness to treatment. It takes the form, as a rule, of a catarrhal ophthalmia, with a coincident eruption of phlyctenulæ and blepharitis, photophobia is a common feature. In addition to this, many outbreaks of measles are ushered in by conjunctival inflammation. During an attack of smallpox, pustules may develop upon the conjunctiva or cornea, and in the latter position may entail serious results. I have occasionally observed small pustules upon the conjunctiva and intermaxillary space during varicella. Apart from acute exanthemata, certain chronic inflammations of the skin may be complicated with conjunctivitis. In acne rosacea a small nodule may form in the limbus, and be accompanied by localised redness and by photophobia. After persisting for several days the inflammatory signs disappear, but a clear, blob-like spot may remain indefinitely. The conjunctiva may be accidentally inoculated with the vaccine virus, although such a result is commoner upon the edge of the eyelids. In a case reported by Cargill, firm, pale, flat swellings in the ocular conjunctiva were found with a persistent form of urticaria, they were noticed to vary in size from day to day. Characteristic conjunctival changes are present in leprosy. Lastly, eczema of the skin or scalp often coexists with phlyctenular affections of the conjunctiva, altogether apart from the pus inoculations mentioned upon an earlier page.

#### TUBERCULOSIS

Tubercle of the conjunctiva is met with clinically under several distinct forms. It is often primary—not associated, as far as one can tell, with tubercle in other parts of the body,—and in that event is almost certainly the outcome of an external infection. This rare affection runs a lingering course, is usually limited to a single eye, and seldom attacks subjects over twenty years of age. It may or may not coexist with lupus of the face or elsewhere. The form most commonly recognised is characterised by the development in the conjunctiva of small yellowish nodules, or of bleeding cockcomb-like granulations, which sooner or later ulcerate. The nodules may be found in any part of the conjunctiva, but the flattened granulations are often confined to that of the lids. The resulting ulcers have ragged edges, grey nodular bottoms, and are often more or less hidden by pus or debris. The eyelids become swollen, heavy, and unsightly, the conjunctiva throws off a mucopurulent secretion, pain or discomfort appears to be more often absent than present. In advanced cases the cornea may become involved. Erosion of the eyelids may occur, and adhesions between the ocular and the palpebral conjunctiva have been seen. The neighbouring lymphatic glands are practically always enlarged and

<sup>1</sup> Preparations containing iodine must be avoided if calomel is being applied, because there is a danger of damaging the conjunctiva if the two come into combination under the form of mercuric iodide.

tender. In another form the disease appears under the guise of a pedunculated tumour, attached by its pedicle to some portion of the palpebral conjunctiva. It resembles a papilloma more than anything else.

**Diagnosis.**—The diagnosis turns upon the discovery of the tubercle bacillus in the discharge or pieces of the diseased conjunctiva. In obscure cases inoculation experiments or the tuberculin test prove useful.

**Treatment.**—An attempt must be made to extirpate the whole of the granulations, as by excision, scraping, or the use of the galvanocautery, after the operation, iodoform should be applied locally. Recurrences should be watched for, and when found, promptly attacked. Internally, creosote and cod-liver oil, separately or combined, appear to be of service. Careful attention must always be paid to the sanitary environment of the patient.

#### SYPHILIS

The initial lesion, or chancre, is now and again observed upon some part of the conjunctiva, especially in children. It forms a round or oval ulcer, the edges of which are usually raised, induration may or may not be present. The corresponding preauricular and angular glands speedily become involved.

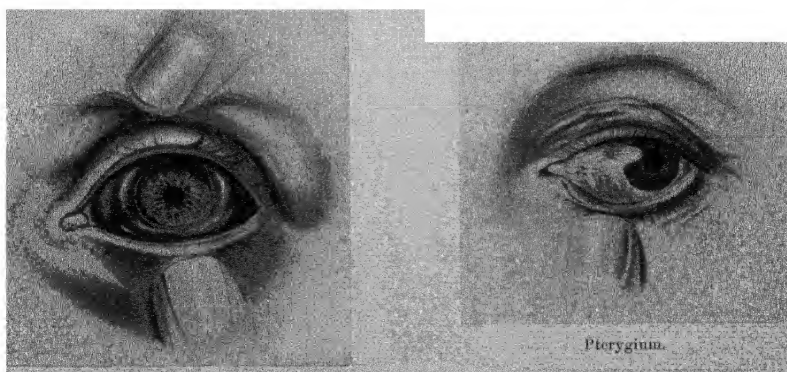
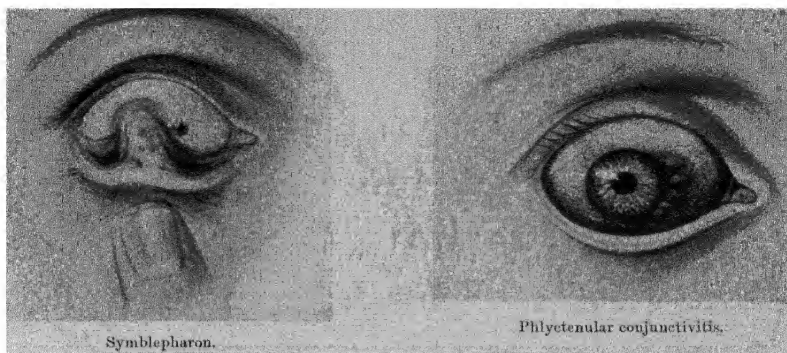
**Treatment.**—The affected mucous membrane should be washed, night and morning, with Lot hydr. nigra (B.P.), and after that the sore dusted with powdered iodoform. As soon as the diagnosis is made, general treatment by small doses of mercury should be begun. Several instances have lately been reported of a curious infiltration of the ocular conjunctiva coming on during the secondary stage of syphilis, rendering the conjunctiva swollen and semi-translucent. **Treatment.**—Mercury internally and black wash locally. Mucous patches have been observed by several writers. Gummata are occasionally found in the ocular conjunctiva, and ulcers may be produced by their disintegration.

**Ophthalmia Nodosa.**—This rare affection is due to the penetration into the eye of the hairs of certain kinds of caterpillars (*Bombyx pin* and *rubra*). It is characterised by many small, hard, greyish growths not only in the lower part of the conjunctiva and sclera, but also in the iris and other parts of the eye. Inflammatory symptoms may attain a high grade. A history may be got of a caterpillar having been thrown against the eye, or the patient, by the nature of his occupation, may have been liable to an injury of that kind. Diagnosis will be rendered certain by excising a nodule for microscopic examination, when it will be found to consist of round and of giant cells, together with a hair. **Treatment.**—The nodules should be removed, and inflammatory symptoms combated by sedative and antiseptic lotions.

#### DEGENERATIONS

**Xerosis Conjunctivæ.**—Xerosis occurs under two forms—the epithelial and the parenchymatous. Epithelial xerosis is a superficial change of the ocular conjunctiva, limited parts of which become dry and lustreless, and are covered with a foam-like, whitish substance, containing the xerosis bacillus in large numbers. The changes are specially prone to affect the temporal side of the ocular conjunctiva, and often take a triangular or oval form. The spots appear slightly raised, and (when magnified) give one the impression of being made up of so many minute globules of mercury. They may be readily wiped away, as with wool, but are reproduced within 24 or 36 hours. The neighbouring conjunctiva may be permeated with dilated vessels, and be thrown into small folds concentric with the edge of the cornea when the eye is moved. This form of xerosis affects mainly thin, poorly-nourished children, who not infrequently suffer from otorrhœa. It makes its appearance in spring or summer, and may recur year after year at that period without entailing any serious mischief. It may or may not be associated with definite night-blindness. It has been shown by the writer to coexist with several other conditions: (1) a deficiency in hæmoglobin, (2) alterations in the visual fields for green and red, and (3) an exaggeration of the fundus reflexes visible with the ophthalmoscope in most young eyes. We assume that epithelial xerosis is the outcome, remotely of lowered nutrition, and immediately of dazzling by bright light. **Treatment.**—The most important remedy is iron, best given in the well-known form of Bland's pill.

**Parenchymatous Xerosis.**—This much more serious malady commonly forms part of some general ailment, the exact nature of which is not well understood. In this country it is found only in young children, who show, besides frothy conjunctival patches, night-blindness and cloudy corneæ. The corneal changes usually go on to perforation, and the patients nearly always die. This malady is generally bilateral, and the usual signs of irritation, such as redness of the eye and photophobia, are often conspicuous by their absence, indeed, the lacrymal secretion may be wholly suppressed. As causes, hereditary syphilis, insufficient or badly-selected food, and prolonged diarrhoea or vomiting, have been mentioned. Among natives in India Herbert found mucous membranes other than the conjunctiva involved—for example, those of the mouth, nose, larynx, bronchi, intestine, and bladder. **Treatment.**—The eyes should be cleansed with sublimate (1:5000) and covered with pads steeped in the same solution. Physostigmine sulphate, gr 1, cocaine hydrochloride, gr 4, distilled water, 1 ounce, may be dropped into the eyes two or



Acute Ophthalmia.

#### DISEASES OF CONJUNCTIVA





three times a day. However, more success is to be looked for from the employment of general than of local measures.

**Pemphigus**—The conjunctiva is occasionally affected by pemphigus, which may or may not be associated with a similar disease of the skin or of other mucous membranes, especially those of the lips, nose, mouth, and fauces. From time to time bullæ make their appearance upon the conjunctiva, which becomes, at first, thick and red, and, later, undergoes cicatricial contraction. It is rare, however, to see the actual blebs, because they are so delicate as to rupture speedily. In this way the conjunctival sinuses are shortened or altogether obliterated, trichiasis or entropion is set up, and the lids become adherent to one another or to the globe of the eye. The corneal epithelium also suffers, becoming dull and dry, like the eye of a dead fish. To this advanced condition the name "essential phthisis" or "essential atrophy of the conjunctiva" has long been applied. The condition is essentially chronic. As a rule, the prognosis is bad, although a few cases seem to recover. According to modern researches, pemphigus is due to a diplococcus, different from any of the ordinary pyococci, and capable of cultivation upon various media outside the human body. The acute form of the disease attacks butchers with disproportionate frequency (Pernet). It seems likely that the conjunctival affection may arise in one of three ways: (1) as a primary disease resulting from octogenous inoculation, (2) as an extension, through the lacrymal passages, from pemphigus of the mouth or nose, and (3) by conveyance of the specific microbe by the patient's finger from cutaneous bullæ. **Treatment**—The treatment is purely palliative. Arsenic may be administered internally, and the conjunctiva kept clean and supple with ointments containing corrosive sublimate (gr  $\frac{1}{2}$ ), iodoform (gr 5), or boric acid (gr 60). Boro-glyceride has also been employed. Attempts to remedy the condition by transplantation of mucous membrane from other parts of the body have so far not yielded very encouraging results.

**Ladaceous Degeneration**—This rare disease has been observed chiefly by Russian surgeons. It is marked by a progressive, non-inflammatory hypertrophy, commencing in the retro-tarsal folds or plica semilunaris, and slowly spreading to other parts of the conjunctiva. The affected mucous membrane is of pale colour, resembles in appearance bacon-fat, and does not bleed when incised. It is said to attack adults, who may or may not have suffered from trachoma, and to occur independently of degeneration elsewhere, as in the liver, spleen, or kidneys. **Treatment**—Excision, partial or complete, is recommended by those who have had experience of the disease.

**Conjunctivitis Peticifera**—Under this name Leber has described a peculiar affection of the conjunctiva, which becomes chronically inflamed and beset with white, opaque dots. The chalky-white colour of the deposits contrasts with the redness of the mucous membrane in which they lie. They gradually get larger, so that the parts involved finally look almost as though they had been petrified. Leber found the deposits to consist of an organic combination of chalk. It is noteworthy that in Leber's case the disease led to partial symblepharon, and that the mucous membrane of the tongue showed epithelial changes.

#### VARIOUS CONDITIONS

**Pterygium**—A condition of the ocular conjunctiva, a fold of which assumes a triangular form, and becomes firmly adherent to the superficial layers of the cornea. The true pterygium occurs only on the inner or outer side of the cornea in the region corresponding to the palpebral fissure. A false pterygium, however, may be met with in other positions. At first, when the pterygium is growing, it is thick and fleshy and marked by obvious vessels, like the wing of an insect; later, it becomes thin and pale and almost of cicatricial appearance. Its course is essentially chronic. During the progressive stage, it gives an unsightly look to the eye, may mechanically limit the ocular movements, and may encroach on the visual part of the cornea. The condition, which is rare in women, is met with chiefly in middle-aged men. It specially affects those who have lived abroad, and is rather common in sailors, probably because they are exposed to vicissitudes of weather. **Therapy**—Air assumes that noxious influence, such as dust, causes the epithelium of the conjunctiva and cornea to be shed, so that a little superadded swelling makes those two structures to adhere and a point or tag of conjunctiva to become fastened to the cornea. Fuchs believes that it originates from a pinguecula which has gradually made its way into the cornea, and in so doing drawn a fold of conjunctiva with it. A kind of spurious pterygium may now and then follow acute ophthalmia, burns, or scalds. **Treatment**—If the condition be stationary, and have not encroached seriously on the cornea, it is best left alone, but otherwise an operation must be performed. The pterygium is carefully dissected away from the cornea, and its apex being folded upon itself, is retained under the ocular conjunctiva by a suture. The conjunctival wound must be closed as neatly as possible by means of a continuous silk thread. The cornea, it must be remembered, always remains cloudy at the spot where the pterygium was attached.

**Symblepharon**—This condition is marked by one or several adhesions between the ocular and the palpebral conjunctiva. It is caused by any-

thing that produces an ulceration of the opposed surfaces, *e.g.* burns, the action of lime, certain forms of acute ophthalmia, tubercle, pemphigus, and operations on the conjunctiva. It affects the lower more frequently than the upper lid. Different names have been applied according to its position, as symblepharon posterius and anterior. The former indicates that the union has involved the conjunctival fornices; the latter that those structures have remained free. Symblepharon totale means that the eyelids are adherent to the eyeball. Symblepharon, when marked, may cause disfigurement, irritability of the eye, limitation of the natural movements, or (rarely) interference with sight. *Treatment*—Many operations, some of a highly ingenious character, have been devised. The simplest plan is to separate the lid from the eyeball by dissection, and then to cover the raw places with morsels of mucous membrane taken from the mouth. Another way is to make liberating incisions in the neighbouring conjunctiva, so that the edges of the wound in the ocular conjunctiva may be brought together with sutures. The treatment of symblepharon posterius is not so satisfactory.

*Pinguecula*—Pinguecula, like pterygium, is seldom seen save in elderly persons. It takes the form of a small yellowish elevation of triangular shape, situated in the ocular conjunctiva, usually on each side of the cornea. The tumour consists of dense connective tissue (which has undergone a hyaline change) covered with thickened epithelium. *Treatment*—Removal, if called for.

*Effusions into the Conjunctiva*—So loose is the texture of the ocular conjunctiva that effusions of blood or serum readily occur into its substance. In extensive hæmorrhages almost the entire ocular conjunctiva may be involved, and blood may even pass for some little distance beneath the epithelial layer of the cornea. It is not uncommon after squint operations for the iris to appear changed in colour, owing to a transudation of extravasated blood into the cornea. The subconjunctival hæmorrhage becomes slowly absorbed. Its main causes are two in number: (1) fragility of the blood-vessels, (2) injury, operation, strain, or inflammation. *Treatment*—Compresses of lead lotion. *Chemosis*, or distension of the conjunctiva by blood serum, may accompany severe inflammations not only of the conjunctiva, but also of other parts of the eye or of its surroundings. It is, therefore, merely a symptom. In a marked case the cornea is overlapped by the swollen mucous membrane, which also protrudes from between the eyelids. Another kind of chemosis, thought to be of non-inflammatory origin, is sometimes observed in elderly persons. Some of the patients suffer from chronic Bright's disease, but, as a rule, the cause of the œdema is obscure. Holmes Spicer has recently described

a form of chemosis, which he considers due to "obstruction of the lymph streams." The patients give a history of acute rheumatism, and the conjunctival affection is associated with lacunar tonsillitis, glandular enlargement, and febrile movement. Lastly, chemosis has been noted by Swan M. Burnet after the internal administration of quinine.

*Staining of the Conjunctiva*—The conjunctiva may be stained by several agents, such as silver nitrate, protargol, largin, and iron sulphate. Lead acetate may lead to local ulceration and to deposition of the salt.

*Lithiasis of the Conjunctiva*—In gouty subjects deposits of uric acid may sometimes be found in the palpebral conjunctiva. Should they cause irritation they may be removed. Small yellowish concretions are quite frequently found in the palpebral conjunctiva of young persons. According to Fuchs, these are to be looked upon as tubular glands of new formation containing fungi (*pilarsen*).

*Conjunctivitis from Dazzling*—A form of painful ophthalmia may quickly follow exposure to snow, electric light, or the X-rays. It is to be treated by cold applications to the lids, and by dropping a 2 per cent solution of cocaine into the conjunctival sac at intervals. It speedily gets well.

*Emphysema*—This implies a communication between the subconjunctival tissue and the nasal cavity or air-sinuses about the orbit. Traumatism is the usual cause. On palpation, characteristic crepitation can be elicited. *Treatment*—Compressive bandage for a few hours.

#### TUMOURS OF THE CONJUNCTIVA

Besides the tumours already mentioned the following new growths have been found in the conjunctiva: cysts, parasitic and otherwise, myxoma, papilloma, fibroma, polyp, epithelioma, sarcoma, and carcinoma. The treatment is to remove the tumour with the least possible disturbance of the neighbouring parts. A simple puncture, however, may sometimes suffice, as when dealing with the so-called "simple cyst." In the case of malignant growths (which generally originate from the limbus) it may be advisable to cauterise the spot of origin.

#### INJURIES OF THE CONJUNCTIVA

The conjunctiva may be injured as the result of direct violence or the action of chemical agents. The degree of inflammation is often out of all proportion to the size of the irritant. Mechanical injuries, doubtless, quickly become complicated with pathogenic organisms, from which the conjunctival sac is seldom free. Moreover, microbes may have been introduced upon the surface of foreign bodies or in other ways. Injuries from lime deserve special mention. They are very formidable when

caused by quicklime, which has a double action, namely, that of heat evolved by contact with the tears and that of chemical irritation. When due to slaked lime, which is more commonly the case, they are less disastrous. *Treatment*.—In severe cases it is necessary to place the patient under the influence of an anæsthetic, and, after removing every particle of foreign matter, to apply castor oil to the injured mucous membrane. Prognosis will always be grave, owing to such secondary results as implication of the cornea, hypopyon, entropion, and symblepharon. Uncomplicated wounds of the conjunctiva heal kindly enough if first made aseptic, and then drawn together by fine silk sutures. It is said that the staining left by grains of gunpowder may be got rid of by electrolysis.

**Conjunctivitis.**—Inflammation of the conjunctival mucous membrane, blennorrhagic, gonorrhæal, croupous, diphtheritic, cecematous, follicular, granular, purulent, membranous, etc. See ADRENAL GLANDS (*Adrenalin, Hay Fever*), CONJUNCTIVA, DISEASES OF, CORNEA (*Phlyctenular Ulcer or Eczematous Conjunctivitis*), GOUT (*Eye*), MEASLES (*Complications, Conjunctivitis*), MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Symptoms, Special Nerves, Eyes*), NERVES, MULTIPLE PERIPHERAL NEURITIS (*Arterial Neuritis, Symptoms*), RHEUMATISM, ACUTE (*Symptoms and Course, Conjunctivitis*), SCLEROTIC, DISEASES OF (*Subconjunctivitis or Episcleritis*), SMALLPOX (*Symptoms, Eruptive Stage*), THYROID GLAND, MEDICAL (*Exophthalmos, Conjunctivitis*), TYPHOID FEVER (*Ocular Complications*), URETHRA, DISEASES (*Gonorrhæa, Complications*).

**Connective Tissues.** See PHYSIOLOGY, TISSUES (*Connective, Mucoid, Fibrous, Cartilage, and Bone*).

**Consanguinity.**—Blood-relationship as opposed to marriage-relationship (*affinity*), of the same blood. There is a widespread belief that marriages of consanguinity predispose to various diseases (albinism, chorea, deaf-mutism, epilepsy, goitre, idiocy, hæmophilia, retinitis pigmentosa, rickets, and malformations) in the offspring, this belief finds expression in the proverb, "Heirathen in's Blut thut selten gut sterben, verderben, oder keine Erben", but there seems to be some doubt whether consanguinity *per se* produces evil effects, whether indeed it is not a bad family history intensified by intermarriage that is to blame.

**Consciousness.** See BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Anæmia, Hæmorrhage, Embolism*), HYSTERIA (*Epileptic Period*), INSANITY, ITS NATURE AND SYMPTOMS (*Relation of Mind and Brain, Biology of Consciousness*), INSANITY, NATURE (*Delusional, Alternate Personality or Double Consciousness*), MEMORY IN

HEALTH AND DISEASE (*Physical Basis of Memory*), UNCONSCIOUSNESS.

**Consensual.**—Reflex actions brought about by sensory impressions without the intervention of the cerebrum itself and its discriminating will.

**Consent.** See MEDICINE, FORENSIC (*Rape*).

**Conservancy System.**—A system of sewage, now generally condemned, by which slop water was got rid of by the drains, while solid excrement was disposed of by means of privies or earth closets. See SEWAGE AND DRAINAGE (*Systems*).

**Conserves.** See CONFECTIONS, PRESCRIBING.

**Consommé.**—A strong broth or soup, for the making of which meat, vegetables, bone, and connective tissue are needed, it forms a jelly when cold. See INVALID FEEDING (*General Preparation of Meats, Soup-Making*).

**Consonants.** See NOSE, EXAMINATION OF (*Character of the Voice*), PHYSIOLOGY, RESPIRATION (*Voice, Consonant Sounds*), STAMMERING.

**Consternatio.**—Pavor nocturnus (night terror) in children, or (in other cases) stupor.

## Constipation.

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See also ABDOMINAL ANEURISM (*Symptoms, Pressure*), BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Paralysis from Vascular Lesions*), BRONCHITIS, ACUTE (*Etiology, Predisposing Causes*), CLIMACTERIC INSANITY (*Treatment of Constipation in*), COLON, DISEASES OF (*Malignant*), GASTRO-INTESTINAL DISORDERS OF INFANCY (*Constipation*), GOUT (*Alimentary System*), HYDROPATHY (*Constipation*), INSANITY, ETIOLOGY OF (*Direct Causes, Sympathetic Insanity*), LUNGS, EMPHYSEMA (*Causes*), MENINGITIS, TUBERCULOUS (*Symptoms*), MYIASIS (*Intestinal*), PALPITATION, PHARMACOLOGY, PREGNANCY, PHYSIOLOGY (*Local Changes*), PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Digestive System*), PURGATIVES, RECTUM, DISEASES (*Persistent Constipation*), TETANY (*Causation*), THERAPEUTICS, HEALTH RESORTS (*Seaside Constipation*), TOXICOLOGY (*Lead*), TRADES, DANGEROUS (*Lead-Poisoning*), VICE (*Alimentary System*).

PHYSIOLOGICAL CONSIDERATIONS.—While the frequency with which the excreta of the bowels are expelled varies at different ages, it may be accepted as practically normal that from later childhood onwards an evacuation at least once

daily should occur. It is true that in the case of many, especially of the female sex, defecation occurs much less frequently—once in three or four days, or even seldom, but while it has to be admitted that many of such cases present apparently few or none of the symptoms of fecal retention, yet none the less such a condition of the excretory functions can hardly be looked on as normal.

The intestinal contents, which in the small bowel are in a very fluid condition, undergo a marked absorption during their passage through the large intestine. Hence it is only in the latter part of the colon that they assume the characteristic semi-solid appearance of a natural motion, and in the sigmoid flexure the feces are stored until the act of defecation takes place. The impetus to this is usually the passage into the rectum (as a result of peristalsis in the sigmoid flexure) of part of the contents of the latter. This is apparently frequently induced by the entrance into the empty stomach of food, especially when the sigmoid flexure is moderately distended with excreta. [An exaggeration of this normal mechanism is seen in what is known as lenteric diarrhoea, where the ingestion of a meal is at once followed by a desire for evacuation as the result of the entrance of a small quantity of feces into the rectum from the merely partially loaded sigmoid.]

Although the act is a reflex one carried out through a centre in the spinal cord, it is largely influenced by the will. In man this centre is situated in the lumbal region of the cord. The presence of the accumulated feces in the rectum causes, by reflex action through the centre, relaxation of the sphincter, while increased intestinal peristalsis is also set up. In voluntary defecation the act is started by a full inspiration, closure of the glottis and fixation of the diaphragm, followed by contraction of the abdominal muscles and levator ani, the latter in this way exerting pressure on the rectum, while the tension of the pelvic fascia and muscles of the pelvic floor offer the required resistance.

**Definition.**—Constipation may be defined as a condition in which the alvine evacuation is difficult and deficient either in amount or frequency, or both, with a consequent tendency to fecal retention.

While in reality largely a symptom present in a great variety of conditions and not a disease, constipation is frequently in itself of so much importance as almost to warrant its being regarded as such. Certainly in view of the great frequency of its occurrence, the amount of discomfort and suffering it produces, as well as the beneficial effects and satisfactory results of its proper treatment, the condition is one which calls for more careful attention and less empirical treatment than it often receives from

the practitioner. (For constipation in children see "Gastro-Intestinal Disorders.")

**CAUSES.**—While the causes of constipation are so numerous and varied that it is difficult to summarise them, it is essential to the satisfactory treatment of any case that the real source of the trouble be discovered. As a rule several factors are at work, and these may perhaps be best considered briefly under two heads—(A) Faulty expulsive mechanism, (B) Faults in the intestinal contents.

**Faulty Expulsive Mechanism.**—In this first group of causes one of the most important is that of *habit*, carelessness in attending to the call to stool, with the result that the bowel becomes accustomed to the presence of fecal accumulations, and one of the chief stimuli to regular defecation is thus lost. In other cases the sedentary habits or occupation of the patient, with the consequent feebleness of the abdominal muscles and sluggishness of the whole circulation, constitute a powerful factor.

In another group of cases the source is to be found in the *bowel*. Apart from the existence of actual constriction from malignant disease in the bowel, the possibility of which must never be lost sight of even in comparatively young subjects, constipation may arise from feebleness of peristaltic force consequent on a lowering of the neuro-muscular tone of the bowel, *e.g.* in old age or anæmia, from cicatrization of old ulcers, from the presence of peritonitis (subacute or chronic) or old-standing bands or adhesions (impeding peristalsis), from pressure on the bowel by tumours or other swellings of adjacent parts, *e.g.* uterine enlargements, physiological or pathological, ovarian tumours, etc., while, again, the over-distension which may result from such blocking of the lumen of the bowel still further tends to perpetuate the malady by lessening the peristaltic force. In other instances, especially of painful pelvic disorders in women, the dread of pain in defecation and actual pain itself may have an inhibitive effect on peristalsis, and the same is seen in cases of hæmorrhoids, anal fissure, etc.

On the other hand, the constipation may arise from an interference with the nervous mechanism concerned in controlling the act of defecation, from involvement of the centres or nerve-fibres, as in some forms of spinal disease, or as a part of various cerebral disorders, *e.g.* melancholia, tubercular meningitis, etc.

Lastly, *inefficiency of the accessory muscular mechanism* may contribute to the production of constipation, as in some cases of paralysis, or in patients whose abdominal muscles have been over-distended, *e.g.* by repeated pregnancies, ascites, etc., or, again, the presence of severe cardiac or pulmonary disease may render any straining efforts on the part of the patient impossible.

**Faults in the Intestinal Contents.**—The solid

as well as the liquid ingesta may be at fault, or, on the other hand, the intestinal secretions may be defective. Thus the food taken may be too soft and too easily absorbed, leaving little or no residue, such a diet in many cases having been adopted because of deficient masticating powers owing to loss of teeth. Or the intestinal contents may be too dry, either from insufficiency of liquids taken or deficiency in the intestinal secretions. Instances of this latter are seen in cases of jaundice, diabetes, etc., or cases of excessive perspiration (from exercise or disease), bringing about increased loss of fluid from the body. In other instances the *quality* of the drinking water is at fault, thus it may contain an excessive amount of lime salts, or it may be contaminated, *e.g.* with lead.

**SYMPTOMS**—When constipation is merely one of the manifestations of the presence of some other malady (*e.g.* tubercular meningitis) its symptoms are scarcely to be separated from those attendant on the primary disease. But while it is undoubtedly true that in many instances of habitual constipation the patient exhibits little in the way of symptoms, yet in the majority of cases there are found to be present to a greater or less extent such manifestations as general languor, torpidity, and depression of spirits, along with irritability, a feeling of fullness in the head, passing into actual headache, furring of the tongue, with foulness of breath and loss of appetite, together with some feeling of fullness and distension in the abdomen, or even pain. In a few cases marked mental disturbance is present. Severe cases may be attended by vomiting, hicough, etc., the usual symptoms of intestinal obstruction.

The characteristic stools in constipation consist of small, dry, hard, often dark, and offensive masses, along with which, in cases of old standing, is occasionally to be found mucus, or even blood, if severe straining has been required for their expulsion.

Lastly, it must never be forgotten that *diarrhoea* may be really a symptom of constipation, the accumulated fecal masses by their presence giving rise to catarrh in the lower part of the bowel, with consequent desire for frequent evacuation. In such cases it is often only on digital examination of the rectum that the existence of constipation is revealed, as shown by the continued presence of scybalous masses in the rectum in spite of frequent evacuations. In addition, what may be termed secondary symptoms of constipation may arise, either locally, *e.g.* prolapse of the bowel, hemorrhoids, etc., or as the result of pressure on the surrounding parts consequent on the loaded condition of the bowel there may be vesical or uterine catarrh, or, again, varicosity of the veins of the leg (especially the left) may be increased, if not actually set up.

Before commencing treatment it is important,

in view of the difference of prognosis in the two conditions, to decide if possible whether the case be one of mere functional derangement or of organic disease.

**TREATMENT**—As has been already insisted on, this must be rational, and adapted to the particular causes at work in each case, and to this end the relatively subordinate position of *drugs* in the treatment of the majority of cases should always be borne in mind. The mere routine ordering of some cathartic remedy, without further attempting to prevent the recurrence of the constipation, cannot be too strongly condemned. The enormous and increasing consumption of innumerable patent medicines indicates only too clearly the extent of the evil, which is unfortunately too often only perpetuated by their injudicious employment. The administration of a vigorous cathartic, however, as a commencement to the thorough treatment of a case of constipation is often not only very beneficial, but even essential, but it is always advisable to explain fully to the patient the general hygienic and dietetic management of the condition before advising the employment of medicinal remedies.

**Habit**—This is one of the most important prophylactic measures in the treatment of constipation. The patient must be urged to go to stool each day *regularly at the same hour*, preferably after a meal, but the time should be chosen with a view rather to its being available *every day* for this purpose. Thus for those whose business requires them to take breakfast at varying hours—often with a hurried rush for trains, etc.—the habit of going to stool at bedtime should be recommended, similarly those who suffer from severe anal troubles, and especially prolapse or piles, should be advised to choose the same hour. Not only will the patient be likely to obtain then greater facilities for sponging himself and returning the protruded mass, but the subsequent rest in bed allows of the congestion subsiding before active exertion is again called for.

It is of the utmost importance that this habit of punctual daily evacuation of the bowels should be insisted on from early childhood in both sexes. Too little attention is paid to this matter, especially in girls, entailing much subsequent annoyance, and even suffering. Those who have not acquired this regular habit must be encouraged to persevere in going regularly to stool at a fixed hour, even although no motion may result at first. In some cases the adoption of the more "crouching" attitude natural to defecation in the open air—as by using a chamber-pot—has been recommended.

Again, with many, smoking after a meal acts decidedly as a laxative, but the all-important point is *regularity and punctuality* in the daily evacuation.

**Diet**—The patient's diet should always be

carefully inquired into. Foods made of the coarser grains, or in the preparation of which part at least of the more indigestible cellulose and fibre is retained, should be substituted for those of the finer sort. "Whole meal" porridge, oatcakes, and brown bread are usually easily taken, and the patient should be encouraged to eat the crust, and not merely the softer parts of the bread. Many people require syrup or treacle to their porridge, and these also are useful laxatives.

Vegetables, by reason of the large proportion of cellulose and neutral salts they contain, are also very helpful, while the gases which some of them are apt to evolve still further stimulate intestinal peristalsis, though they may somewhat increase the patient's flatulent distension. Thus raw tomatoes, salad, or merely fresh lettuce should be recommended, and as a general rule the various vegetables in season should enter largely into the regular dietary.

Similarly fruit, raw or cooked, should be utilised freely. An orange or a raw apple before breakfast is an efficient laxative to many, and the patient should be advised to see that fruit always forms a part of his daily dietary. The attraction which soft (non-laxative) milky foods have for those whose teeth have failed them must be counteracted by combining with these such fruits as stewed figs or prunes, apricots, etc., and if the fibres of these prove too tough for the patient's imperfect teeth artificial ones must be obtained. Fruits in the form of jams also are of advantage, and marmalade possesses marked laxative properties. Milk and milky foods should be sparingly used, and of these latter the coarser-grained varieties, e.g. sago or whole rice, are preferable to such as arrowroot and cornflour.

**Liquids**—The importance of attending to the question of the amount of liquid consumed by the patient can hardly be overestimated. Many people, especially women, seldom take a drink of water, tea—too often with a fairly strong proportion of astringent tannin in it—being almost their only beverage. A single tumblerful of water taken on an empty stomach on rising—or, if required, at bedtime also—will frequently, in conjunction with the other general directions mentioned, have the desired effect. If the patient perspires much he should be recommended to drink water still more freely, especially between meals. Excessive "hardness" of the drinking water from the presence of lime-salts must be remedied by boiling the water and filtering, or by the use of rain-water for cooking purposes as well as drinking.

Coffee or cocoa is preferable to tea, containing less of the astringent tannin, and malt liquors tend to be more laxative than spirituous liquors. Buttermilk may also be ordered as being more laxative than ordinary milk.

**Exercise**—The frequency of constipation in those of sedentary habits is well known, but the

mere ordering of exercise is not enough. Care must be taken to see that the patient adopts some form of exercise likely to have a beneficial effect upon his abdominal muscles, and so on the underlying intestines. Thus riding on horse-back, cycling, golfing, tennis, and swimming are much more likely to benefit than merely walking, while special gymnastic exercises calculated to strengthen the abdominal muscles (often so feeble in females) are of the utmost value.

One precaution, however, must be borne in mind. Should exercise, as in the case of some delicate women with pelvic disorders, give rise to pain, the constipation will probably be rather increased than diminished, the irritation inhibiting peristalsis apparently reflexly through the splanchnics. In such cases even moderate exercise may have to be prohibited, and (as afterwards referred to) an opiate may be required as a laxative.

**Massage, etc.**—This is frequently of most service in those cases in which the beneficial forms of exercise cannot be carried out, viz the elderly or the very young. Especially useful is regular systematic massage of the colon in the direction of peristalsis, carried out by the patient himself or a trained attendant. Others have strongly recommended *vibration* as being a more potent and less dangerous mode of stimulating peristalsis in severe atonic cases. With this may be combined the use of cold compresses or cold douches to the abdomen, while in some obstinate cases beneficial results are obtained from the regular use of *electricity* to restore tone to the intestinal walls as well as the abdominal muscles. For this, special forms of electrical apparatus have been devised.

**Medicinal Remedies**—Should the various hygienic and dietetic measures above referred to fail to effect a cure of the condition, recourse must be had to medicinal remedies, but only as an *adjunct*.

The number of cathartic or laxative remedies is enormous, yet only a few salient points will be referred to.

(a) **Enemas**—Where the constipation is of long standing, with an accumulation of hard scybalous masses in the rectum and sigmoid flexure, these should be softened by enemas of soapy water or olive oil and oxgall, while in severe cases it is sometimes necessary to break down the masses and remove them by means of a spoon handle, care being taken to see that the accumulation is thus thoroughly removed. Should some scybalous masses be lodged higher up in the intestine, large douches of warm water inserted by means of a long tube connected with a head of water should be persistently employed. It is well to remember that some writers have pointed out that, once this form of treatment has been begun, there is a risk of auto-intoxication—apparently from the liberation of toxins from the softened scybala—if the douches are not

administered *daily* so long as any masses remain in the bowel. Again, as a stimulant to defecation small enemata of glycerine, or suppositories of the same, prove very useful, the forms of the latter now obtainable constituting one of the most convenient and least injurious therapeutic remedies we possess. Especially convenient are they for patients who are from home or traveling, but the habit of employing them systematically is to be guarded against. Occasionally considerable benefit may be derived from the use of *small* enemata of *cold* water, which appear to act by stimulating peristalsis.

(b) *Drugs*.—All cathartic remedies which have an *astringent* tendency as part of their after-effects should be rigidly avoided in treating habitual constipation, *e.g.* Gregory's powder. What should be aimed at in prescribing is to give something which may so tone up the bowel to act that the drug can be dispensed with in a short time. Especially important among these intestinal tonics are cascara sagrada, nut vomica, or strychnine, and aloes. These may be given together conveniently in pill form, a little belladonna being often added with or without hyoscyamus to prevent griping. It is most important to impress on the patient that it is not a *purgative* pill he is getting, but a *tonic* for his intestines, and therefore he is not to expect an action of the bowels as the result of one or two pills. Such a pill as—*R* Extr cascara sagrada, gr i ss, extr nucis vomica, gr  $\frac{1}{4}$ , extr aloes, gr  $\frac{1}{2}$ , extr belladonnae, gr  $\frac{1}{8}$ , ext gent co qss, given twice or thrice daily, *always as an adjuvant to the general measures stated previously*, seldom fails to bring about in a few days a regularity in the evacuations which continues after the pills have been *gradually* omitted. In anæmic females the addition of a little iron in some form to the pill is often of great advantage. Again, the liquid form of cascara (with which an equal amount of glycerine may be advantageously combined) is sometimes very effectual if taken nightly for some time to tone up the intestine, always in addition to dietetic and other general measures. The patient should be always strongly warned against indulging in occasional purgatives, and encouraged to persevere in measures calculated to bring about a natural daily evacuation.

Sometimes in plethoric subjects, or where the motions are excessively dry, salines given in the morning act best, either in the form of one of the numerous aperient waters, *e.g.* Hunyadi Janos, Rubinat, etc., or as the simple salts, *e.g.* Carlsbad, etc., but while often beneficial for a time, and especially useful in obstinate cases as an alternative, *e.g.* during summer weather, they are never of the same lasting benefit as the purely intestinal neuro-muscular tonics, and if persevered in are apt to perpetuate the condition they were designed to cure. The advantage of sending cases of habitual constipation to one

of the many purgative springs is doubtful. So far as the *cure* of his constipation is concerned the really important part of the treatment lies in his acquiring there those habits of regular hours, punctuality in attending to the call to stool, systematic exercise, etc., which he can follow out equally well at home. The action of the purgative waters is too often followed only by a recurrence of the constipation on their cessation.

Again, when there is sluggishness of the liver present, hepatic stimulants, *e.g.* euonymin, or podophyllin, are called for, but such drugs must be adopted as mere temporary additions to the systematic treatment of the constipation in the manner stated above.

Lastly, in some obstinate cases of constipation, as previously referred to, an opiate (often combined with belladonna) will open the bowels when various cathartics have failed, apparently by allaying the reflex inhibition of peristalsis, and in a similar fashion an opiate may be advantageously combined with a purgative if spasm or pain be a prominent feature in the case.

**Constituents.**—The vehicle or excipient in a prescription, giving consistence, or helping to remove disagreeable tastes or smells. *See* PRESCRIBING.

**Constitution.**—The particular way in which an individual reacts to external circumstances, and resists or yields to morbid influences, predisposition to develop special forms of disease, *e.g.* nervous, rheumatic, gouty, and the like, temperament. Among the constitutions or temperaments which used to be much dwelt upon were the nervous, the phlegmatic, the bilious, the sanguine, the gouty, etc.

**Constitutional Diseases.**—In a somewhat loose fashion it is permissible to speak of certain diseases, *e.g.* gout, syphilis, rheumatism, tubercle, rickets, rheumatoid arthritis, acute and chronic alcoholism, diabetes mellitus, hæmophilia, obesity, and some nervous maladies, as being constitutional, *i.e.* developed as the result of certain inherited or acquired morbid modes of action of the tissues and organs. *See* under the above-named diseases.

**Constrictors.**—Muscles which diminish the calibre of vessels or the capacity of hollow organs by their contraction, *e.g.* the constrictors of the nares, of the vagina, of the bladder, and of the pharynx (*see* PHARYNX, AFFECTIONS, *Neuroses*).

**Consultation.** *See* ETIQUETTE, MEDICAL.—The deliberative consideration of a medical or surgical case in which the diagnosis, or prognosis, or treatment is not clearly indicated, or is not thought to be so by the patient or his friends, with a view to greater accuracy and benefit, two or more practitioners may take part.

**Consumption.**—A wasting away, especially that due to tubercular disease of the lungs, *galloping consumption* is the name applied to the more rapidly advancing form of phthisis. See GASTRO-INTESTINAL DISORDERS OF INFANCY (*Chronic Diarrhea, Consumptive Bowels*), LUNG, TUBERCULOSIS OF.

**Contagion.**—This word (contagion), from the Latin *contingo*, I touch, is often used almost as a synonym of *infection*, but, strictly speaking, it means the communication of a disease from one person to another, directly (immediate) or indirectly (mediate, or by a third person), by means of a "particulate or sensible material" or "contagium", the same disease is produced in the second person as in the first, and it also has the same power of being passed on, by contagion, to another person. See TYPHUS FEVER (*Etiology*).

**Continued Fever.**—A now little used name for a fever in which the temperature varies little during the twenty-four hours, but which steadily progresses (without intermissions) towards its height, and thereafter slowly declines, or may, when at its height, terminate fatally, it was applied to relapsing, typhus, typhoid, and other fevers, such terms as *simple continued*, *malignant continued*, *marasmic continued*, and *ardent continued* fever are practically obsolete. See TROPICS, UNCLASSIFIED FEVERS OF (*Continued*).

**Contracted Kidney.** See NEPHRITIS (*Renal Cirrhosis*).

**Contraction.** See PHYSIOLOGY, TISSUES (*Muscle, Physical Characters, Contraction*), LABOUR, RETENTION OF PLACENTA (*How-glass Contraction of Uterus*), LABOUR, PROLONGED (*Pelvic Deformities, Contraction*).

**Contracture.**—Permanent contraction (e.g. of a muscle) with rigidity, after convulsions, paralysis, or rheumatism, it is sometimes distinguished as hysterical, myopathic, neuro-pathic, or paralytic. See BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Paralysis, Rigidity*), BRAIN, TUMOURS OF (*Symptoms, Tonic Spasm*), BRAIN, CYSTS OF (*Porencephaly*), FACIAL NERVE, PARALYSIS OF (*Symptoms, Contracture*), HIP-JOINT, DISEASES OF (*Neuro-Arthropathies*), HYSTERIA (*Motor Disorders, Paralysis and Contractures*), HYSTERIA, SURGICAL ASPECTS OF (*Hysterical Contractures*), JOINTS, DISEASES OF (*Impaired Mobility, Contracture*), KNEE-JOINT, DISEASES OF (*Deformities following Disease, Contracture*), MALINGERING (*Contractures*), TETANY (*Synonyms and History*).

**Contraindication.**—The condition or state of the patient which indicates that some drug or some method of diagnosis or treatment cannot safely or with benefit be employed.

**Contre-Coup.**—The effect of a blow (e.g. a contusion of brain substance or a fracture of a bone), produced either exactly opposite to or at a considerable distance from the site of the stroke. See BRAIN, SURGERY OF (*Contusion*).

**Contrexeville.** See BALNEOLOGY (*France, Calcareous*), MINERAL WATERS (*Earthly and Calcareous*).

**Control.** See PHYSIOGNOMY AND EXPRESSION (*Expression of Brain Faculty, Control*).

**Control Experiment.**—An experiment made simultaneously with another, in which all the conditions, save one, are the same, a check or test experiment.

## Contusions.

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**DEFINITION.**—A contusion may be defined as a traumatic laceration of the subcutaneous soft tissues of a part, without solution in the continuity of the skin. When the integument gives way a *contused wound* results.

**CAUSES.**—The force producing a contusion is always applied by a *blunt* object, is momentary in its action, and immediate in its effects. It may be in the form of a direct blow acting perpendicularly to the surface, as in the case of a stroke with a stick, or a blow with the closed fist, or the force may impinge obliquely, as when a spent shot glances off the body, or a carriage wheel grazes a limb in passing. The damage resulting from a "percussing" force acting at right angles to a part is more or less localised to the seat of impact, but may extend deeply, while the effects of an obliquely directed stroke tend to be more diffuse and superficial. At the same time there may be much tearing of the subcutaneous tissues from the latter form of violence.

On the other hand, a part may be contused by "pressure," as when a limb is squeezed between buffers, or crushed by a waggon wheel passing over it. The resulting lesions are more widespread and deeper than in the case of injuries by percussion.

**MORBID ANATOMY.**—Three degrees in severity of contusions are recognised.

In the *first degree* or *contusion with ecchymosis* there are small, interstitial, petechial hemorrhages resulting from the rupture of minute blood-vessels in the skin, with slight oedema of the whole of the injured part. The effused blood is as a rule spontaneously and rapidly reabsorbed.

*Second degree* or *contusion with extravasation*



*of blood*—The extent and character of the effusion in contusions of the second degree depend upon the looseness or density of the connective tissue of the injured part, the size and nature of the damaged vessels, and the degree of force employed to produce it.

It may be that all the tissues are infiltrated with blood from a number of torn veins and small arteries, or that the blood from a ruptured artery or large vein collects in a single space, constituting a *hematoma*.

In either case the bleeding is soon arrested by the gradually increasing pressure in the effusion, and by the coagulation of blood in the torn vessels. The extravasated blood tends slowly to diffuse itself along the lines of least resistance—under fascia, between muscles, into vascular sheaths or connective-tissue spaces—so that it may eventually reach the surface at some distance from the seat of injury. This fact is sometimes of importance in the diagnosis of injuries, especially in certain fractures of the base of the skull, where the ecchymosis appears after some days under the conjunctiva, or behind the mastoid process.

The majority of contusions tend to spontaneous cure. The fluid part of the extravasated blood is reabsorbed by the lymphatics. Some of the red corpuscles regain the general circulation directly or by the lymph stream. The solid clot disintegrates. Hematoidin crystals are precipitated in the tissues, giving them a bluish, green, or yellow colour, and crystals of cholesterol may form from destruction of the fatty elements of the clot. The remaining debris is carried off by the lymphatics, and may cause temporary enlargement and pigmentation of the adjacent lymphatic glands.

The destroyed tissue is replaced by a cicatrix formed by a process analogous to that known as healing by the first intention. It is not uncommon for proliferation of the connective tissue surrounding an effusion of blood to take place to such an extent that an elementary membranous capsule is formed. In this way a *hemorrhagic cyst* is developed, the contents of which may in course of time become absorbed, or, after drying up, undergo calcareous degeneration.

Certain contusions result in the outpouring of a large quantity of *serous fluid* instead of blood, most probably from tearing of large lymphatic vessels. This is commonest after obliquely directed blows or crushes, acting chiefly on the superficial structures, especially when a tense fascia underlies the skin. Such effusions are slowly reabsorbed, sometimes remaining unchanged for months.

In a few rare cases, especially where the contused tissue contains much fat, or where a fracture coexists, localised collections of an oily fluid form, and are slowly reabsorbed.

*Third degree or contusions with complete dis-*

*integration of tissue*—When the vulnerant force is extreme, and the resistance of the contused tissues slight, their structure may be completely destroyed. Muscular tissue, blood-vessels, nerves, and connective tissues are reduced to a pulp, the circulation through the part is suspended, and the vitality so depressed that necrosis takes place. The overlying skin becomes cold, livid, and dark-coloured, and blisters containing serous or bloody fluid may form on the surface. If septic infection be prevented dry gangrene takes place, but if bacteria gain access suppuration and moist gangrene ensue.

**CLINICAL FEATURES**—The chief local phenomena of contusions are discoloration and swelling. When the condition is of the *first degree* minute punctate hemorrhages are seen scattered through the superficial layers of the skin over the affected area. These are separate from one another, of a light colour, and with slight edematous swelling around them.

Contusions of the *second degree* are those most commonly met with. The effused blood occurs in patches varying in size and depth with the degree of force which produced them, and in shape with the instrument employed. Where the skin is naturally thin or pigmented, as over the inner aspects of the arm, in the eyelids, scrotum, and penis, the discoloration tends to be darker than elsewhere. When the extravasated blood is only separated from the oxygen of the air by a thin layer of epidermis or by mucous membrane, it retains its bright arterial colour. This is often well illustrated in cases of black-eye, where the blood effused under the conjunctiva is bright red, while that in the eyelids is almost black.

The discoloration may take some days to appear on the surface if the primary effusion has been deep-seated among the muscles and under strong fascial bands, and in these circumstances it may appear some distance from the seat of injury. The presence of blood extravasated deeply in the tissues can, however, often be detected by the firm, resistant, doughy swelling which exists. On deep palpation a peculiar sensation, closely simulating the crepitus of fractures, is sometimes transmitted to the fingers, and is liable to lead to errors in diagnosis.

The changes which take place in the effused blood lead to characteristic alterations in the colour of the contused part. In from 18 to 24 hours the margins of the blue area become of a violet hue, and as time goes on the discoloured area increases in size, and becomes successively green, yellow, and lemon-coloured at its margins, the central part being the last to change its hue. The rate at which this play of colours proceeds varies so much, and depends on so many circumstances, that no time limit can be laid down. A large bruise over the arm of a healthy person may disappear completely in two or three weeks,

while one of corresponding size and severity over the pelvis of a weakly patient will persist for as many months

In all contusions of the second degree there is marked *swelling* of the whole area involved, especially when the subcutaneous areolar tissue is abundant and open, as in the eyelids, scrotum, penis, and labia

In contusions of superficial parts there is always at first great *pain* and tenderness on pressure, but it soon passes off. When dense fasciae, ligaments, or periosteum are involved these symptoms are more severe and lasting. *Traumatic neuralgia* along the course of a bruised nerve-trunk is not uncommon, and marked *hyperaesthesia* over a bruised area frequently persists for a long time. Although the degree of *shock* is not always proportionate to the severity of the injury, sudden *syncope* frequently results from severe bruises of the testicle, abdomen, or head, and occasionally marked *nervous depression* follows these injuries. The function of a bruised part is always seriously interfered with while the effusion and swelling last. The only evidence of general *constitutional disturbance* is a temporary elevation of temperature to 102° F, or even higher—a form of waste-product fever. Among the rarer clinical features may be mentioned *general anaemia*, when the local effusion of blood is very great, *traumatic icterus*, when the blood pigment is deposited throughout the skin of the body, and *fat embolus*, which is usually associated with a complicating fracture.

The clinical features of contusions of the *third degree* are of secondary importance to those of the graver injuries with which they are usually associated—fractures, dislocations, laceration of large vessels, nerves, or muscles—conditions which in diagnosis and treatment overshadow the accompanying contusion.

**TREATMENT**—The main indications are (1) to prevent the further effusion of blood, (2) to alleviate the pain, (3) to maintain the vitality of the damaged tissues, (4) to promote the absorption of the blood and lymph already extravasated, (5) to avoid, or (6) to combat, bacterial infection.

If seen immediately after the accident the part should be placed at absolute rest in a slightly elevated position. Firm elastic pressure through a thick pad of cotton-wool is of great value in arresting the effusion, and cold (in the form of ice), lead and opium lotion, or a weak carbolic compress, are useful adjuncts.

When extravasation has already taken place massage is the speediest and best method of dispersing the effused products. The part is deeply kneaded and rubbed in a centripetal direction once or twice daily, and the patient is encouraged to move the part freely after each sitting, in order that the muscular movements may augment the action of the rubbing. Any

abrasion of the skin of course contraindicates the employment of massage.

When the effusion is so large and so tense as to threaten the vitality of the tissues an incision may be indicated, but is only to be practised when the certainty of maintaining asepsis is assured.

Serious complications will be met on general principles by incision and drainage, or, if necessary, by amputation.

**MEDICO-LEGAL ASPECTS OF CONTUSIONS**—Contusions have often very important medico-legal bearings. When called to see a suspicious case the practitioner should note (1) the date when the bruise is alleged to have been inflicted, (2) the date of the examination, (3) the degree of the contusion and the precise colours exhibited at the time of examination, as an indication of the approximate age of the ecchymosis, (4) the shape of the discoloured area, in association with the character of the instrument with which it is alleged to have been produced, (5) the presence or absence of evidence of such complications as fracture, dislocation, external wounds, or injuries to internal organs.

It is well to bear in mind that those suffering from scurvy, haemophilia (bleeders), and fat, anemic persons in a low state of health may have extensive ecchymoses produced on their bodies by very trivial injuries, and further that ecchymosis may occur, apart from external injury, from violent muscular efforts, as in attempting to recover one's balance, or during severe vomiting or other form of straining. It is also worthy of note that many old people with weak circulation have darkly discoloured patches on the legs and feet which closely resemble contusions, and might be mistaken for them on the cadaver. The necessity for distinguishing between bruising and post-mortem lividity need only be mentioned.

On the other hand, the absence of ecchymosis does not prove that no external violence has been inflicted, as severe blows, especially over the abdomen and thorax, are often not followed by external signs of bruising, even although internal organs are seriously damaged.

Violence inflicted on a living body may not be manifested by ecchymosis till after death, while blows dealt on a recently dead body (within two hours of death) may produce signs exactly like those occurring on the living. If, however, the body be cold and cadaveric rigidity has set in, the appearances of contusions on the living cannot be produced by external violence.

If a bruise on a dead body be found uniformly blue or livid, the presumption is that the blow was struck immediately before death, while a play of colours round the margin suggests that some time has elapsed between the infliction of the injury and death. That the effused blood has remained fluid is not proof that the contusion was sustained after death.

Evidence of the previous existence of a bruise may be found for some time in the pigmentation of the neighbouring lymphatic glands

**Conus Arteriosus.**—The conical prolongation of the upper and left angle of the right ventricle of the heart, the infundibulum, its stenosis constitutes one of the varieties of congenital heart disease. See HEART, CONGENITAL MALFORMATIONS OF

**Conus Medullaris.**—The conical termination of the spinal cord which lies immediately above the slender *filum terminale*, it has been injured during lumbar puncture

**Convalescence.**—The period of gradual restoration to health after an illness. See INVALID FEEDING (*Diet during Convalescence*), PUERPERIUM, PHYSIOLOGY (*Management*), TYPHOID FEVER, etc

**Convallaria Majalis.**—The leaves, stem, and flowers of the lily of the valley constitute the non-official medicine (*Convallaria Majalis*), *convallamarin* ( $C_{22}H_{44}O_{12}$ ) is the active principle, and the plant contains also *convallarin* ( $C_{24}H_{40}O_{11}$ ), both of them glucosides, the *Tinctura Convallaria* (made from the flowers) is given in doses of 5 to 20 m in the same class of case as digitalis. See PHARMACOLOGY, DIGITALIS

**Convergence.** See ACCOMMODATION (*Accommodation and Convergence*), BRAIN, PHYSIOLOGY OF (*Third Nerve, Median Nucleus*), EYE, CLINICAL EXAMINATION OF (*Visual Acuity, Power of Convergence*), OCULAR MUSCLES, AFFECTIONS OF (*Paralysis, Squint*), STRABISMUS

**Convolutions of the Brain.** See BRAIN, PHYSIOLOGY, BROCA'S CONVOLUTION, GYRUS, PHYSIOLOGY, NERVOUS SYSTEM (*Cerebrum*)

**Convulsions.**—Violent irregular motion of the whole body or parts of it due to involuntary contractions and relaxations of the voluntary muscles, different varieties are apoplectic, cataleptic, choreic, clonic, eclamptic, epileptic, epileptiform, hysterical, infantile, intertinal, mimetic, oscillatory, puerperal, salarum, tetanic, tonic, and uremic. See ANEURYSM (*Common Carotid, Treatment by Ligature*), ANTISPASMODICS, BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Anæmia, Edema*), BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Thrombosis, Vascular Lesions and their Results*), BRAIN, INFLAMMATIONS (*Clinical Features*), BRAIN, TUMOURS OF, BRAIN, CEREBELLUM, AFFECTIONS OF (*Tumour*), CONVULSIONS, INFANTILE, GASTRO-INTESTINAL DISORDERS OF INFANCY (*Chronic Diarrhoea, Complications*), GENERAL PARALYSIS (*Diagnosis*), HYSTERIA, LABOUR, OPERATIONS (*Forceps*), MEASLES (*Convulsions*), MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Symptoms*), MENTAL DEFICIENCY, NOSE, POST-

NASAL ADENOIDS, PARALYSIS (*Cerebral Diplegia, Infantile Hemiplegia*), PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Nervous System, Convulsions*), SYPHILIS (*Children, Nervous System*), TOXICOLOGY (*Lead*), TRADES, DANGEROUS (*Lead-Poisoning*), UNCONSCIOUSNESS, UREMIA

## Convulsions, Infantile.

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See also CONVULSIONS

**DEFINITION.**—The term convulsions is applied to more or less general purposeless muscular contractions, occurring simultaneously and successively for a variable time, attended in most instances by a loss of consciousness more or less profound. The more strictly local muscular contractions are more appropriately and conveniently denoted as spasms

**FREQUENCY.**—The frequency of convulsions in infancy has been greatly exaggerated, especially amongst the lay public

The convulsive movements of the limbs seen in moribund children, mainly due to the venous state of the blood, must not be considered in the same category

**ETIOLOGY.**—The causes of convulsions can be divided into predisposing and exciting, of which the former are the more important

**Predisposing Causes.**—1 All writers seem inclined to place as foremost amongst the predisposing causes what has been termed the *instability or irritability* of the infantile nervous system. This instability disappears with advancing age and the further development of the higher centres

2 An inherited neurotic tendency or constitution is a very important factor. Published statistics give a neurotic history in 32 per cent of the parents, and if convulsions in brothers and sisters of the patients are considered, then the family neurotic history rises as high as 67 per cent. The importance of this factor in the causation of convulsions has not as yet been sufficiently recognised and acknowledged

3 Rickets is a further predisposing cause. Whilst, however, a very large percentage of convulsive infants are rachitic, yet convulsions do not occur in certainly more than 5 per cent of all cases of rickets. When convulsions occur in this last they are frequently associated with laryngismus stridulus and tetany. The explanation of the occurrence of all three disorders is, perhaps, due to the brain suffering in the general malnutrition with the rest of the body in rickets

**Exciting Causes.**—Almost any central or peripheral lesion in infancy may act as an exciting cause, especially in subjects predis-

posed. The more common conditions may be thus classified:—

1. Local disease or injury of the brain or its membranes, *e.g.* intracranial tumour, hemorrhage, meningitis, etc. The symptoms of cerebral tumour in young children often present themselves with a startling abruptness, and convulsions at the onset may be general in character. If the convulsions date from birth they are generally due to meningeal hemorrhage, and are often associated with paralysis and subsequent amentia. In not a few cases of meningitis, convulsions may be the first prominent symptom, and such convulsions may be unilateral in character.

2. Altered vascular states, as anemia, following hemorrhage or exhausting disease from any cause, venous engorgement, or, more rarely, uremia.

3. The onset of the acute infectious diseases, *e.g.* pneumonia, etc. The frequency of this relationship has been largely overestimated, convulsions being much more frequent in the course of such affections, and then usually dependent on the venous state of the blood (asphyxia).

4. The developmental conditions resulting in idiocy.

5. Peripheral nerve irritation. Teething disorders and gastro-intestinal derangements are important factors in predisposed subjects. Severe surgical injuries, such as extensive burns, may certainly cause convulsions in infants in whom no predisposition is to be found.

6. Asphyxia. The important part played by asphyxia is perhaps not duly appreciated by the profession generally. This association clinically between asphyxia and convulsions is in strict accord with physiological experiments. Illustrative examples have already been given. The convulsive movements seen in various moribund states are probably also of similar origin.

**PATHOLOGY**—The real pathology of convulsions is as yet undetermined. The post-mortem changes found in the brain and cord are in all probability results and not causes of the disorder. Probably the ultimate pathological processes concerned will be found in molecular changes in the nerve cells, rather than in changes capable of demonstration with any means yet at our disposal. When the pathology of idiopathic epilepsy is revealed to us, the discovery of that of infantile convulsions will be close at hand.

**SYMPTOMS**—The phenomena of a typical attack of convulsions are identical in character, time, and sequence with those occurring in an epileptic fit, and require no detailed description. In both disorders the attacks generally commence with a dazed or "far-away" expression of the face, immediately followed by momentary pallor and dilatation of the pupils. These are closely followed by loss of consciousness, during which the muscles are affected at first with

tonic spasms and apnoea, succeeded after a variable interval by clonic contractions. In both convulsions and epilepsy there is frothing at the mouth, blood-stained if the tongue be bitten, and in both there may be involuntary discharge of urine and feces. The attacks always conclude with a period of drowsiness more or less profound. Several of the phenomena may be but slightly prominent or even omitted, and convulsions, like epilepsy, may vary in their intensity between the widest extremes. A conspicuous feature of infantile convulsive attacks is the well-known turning in of the thumbs upon the palms of the hands, with flexion of the fingers around the thumbs. Convulsions may come on suddenly in the midst of apparently perfect health. In other cases there may be warnings in the shape of fretfulness, squinting, grinding of the teeth, and turnings in of the thumbs upon the palms of the hands.

**DIAGNOSIS**—If the child be seen in the attack the diagnosis can present no possible difficulty. When it has to be made from the verbal accounts of the parents or friends, then the utmost caution and discrimination will have to be used. The too ready acceptance, without careful inquiry, of the lady's interpretation of what constitutes convulsions, has helped to spread and perpetuate an exaggerated impression of the frequency of convulsions proper even amongst the members of the medical profession.

**Prognosis**—The immediate prognosis is generally favourable. When the attacks so rapidly follow one another as to be merged into one long series (status convulsivus), then death may ensue from exhaustion. This last condition—"status convulsivus"—differs in no respect from the "status epilepticus" of adults, except that in the younger subjects the temperature runs much higher and generally reaches 105° or upwards. Death, too, is not very infrequent in convulsions associated with laryngismus stridulus, but here the last-named is possibly more often responsible for the fatal issue than the concomitant convulsions. Convulsions arising during whooping-cough are often fatal, more especially if the infant's health has been much reduced by vomiting or other complication. When broncho-pneumonia is marked by urgent dyspnoea, convulsions, due to asphyxia, are not infrequent and are generally fatal, but the conditions preceding them in this instance are of extreme gravity independently of them.

In most cases the *remote prognosis* of an attack of convulsions should be a guarded one. There is seldom anything in the phenomena of the attacks that furnishes any criteria enabling one to discriminate between convulsions likely to be followed by later neurotic manifestations, and those that are of merely temporary import. Prognosis then must depend less on the phenomena of the attacks than on *their history and*

the circumstances which attend them. Here, however, it may be said at once that the same greater significance attaches to unilateral convulsive attacks in all cases in infancy as to those occurring in later life. Favourable factors are a history of previous good health in the infant, and the presence of some well-defined exciting cause, such as the onset of one of the exanthemata or croupous pneumonia. The prognosis is favourable, too, in cases where the convulsions can be shown to have mainly depended upon asphyxia, the cause of which has been removed or recovered from. Where there is a family neurotic history the prognosis should be guarded in the extreme. Caution should be used, too, in pronouncing as to the future of an infant who has suffered from convulsions presumably from such slight causes as dentition, bowel disturbance, or ascariasis, for it may well be asked whether a nervous system that has once broken down under such slight causes would not be likely to do so again in the future at any times of cerebral stress or strain beyond the ordinary convulsions occurring in the course, not at the onset of scarlet fever, and which are independent of any kidney involvement, are often unilateral at the commencement, and are frequently continued into later life as epilepsy. When the convulsions occur in association with rickets, improvement in the last complaint generally leads to the final disappearance of any disturbance of the nervous system. But even here Sir William Gowers has shown that 10 per cent of adult epileptics have their start in infantile convulsions due to rickets, and in juvenile epileptics the proportion is probably greater. This should lead to a more guarded prognosis than is usually given in cases of rachitic convulsions. Cases where convulsions recur from time to time without organic cause are to be regarded in the most serious light, a large proportion of them merging into epilepsy in the near future, while others are probably the subjects of other neuroses in later life.

**TREATMENT — During the Attack** — In the majority of instances it is only necessary to loosen the clothing about the neck, chest, and abdomen, and to lay the infant in the supine position with the head slightly raised. The infant can then be left to recover from the drowsiness naturally following, or forming part of, the attack. The ordinary domestic remedy, placing the child in hot bath, does no harm probably if the child's health be not too enfeebled. In this last condition, instead of a plain hot water bath, a mustard one might be tried with advantage for the sake of its well-known stimulating properties. There is nothing to be said in favour of the other routine remedy, the application of cold to the head, except in the cases where it is used for the express purpose of lowering the temperature. If the unconsciousness be unduly profound, and especially if it be

attended with other fits, or threatenings of them, then recourse must be had to more active measures. First amongst these is the *inhalation of chloroform*, which is warmly advocated by Heneoch, John Thomson, and other authorities. Profound unconsciousness is no bar to the use of the drug, and usually recovery from the anæsthetic is accompanied by the return of consciousness. Eustace Smith praises the hypodermic injection of morphia in these cases in doses of  $\frac{1}{4}$  of a grain to an infant of six months old, and says it can be safely repeated, if necessary, in the course of half an hour. The inhalation of *nitrite of amyl* in one-gram doses has been recommended by some authorities. The administration of *chloral* by enema, in doses of about three grains to an infant of six months old, is a valuable remedy for two reasons. If the drug be retained its sedative action on the nervous system is usually quickly manifested. In other cases its presence in the rectum leads to the evacuation of an especially foul-smelling motion, and this is generally followed by a speedy return of consciousness. If there be any valid reason for suspecting indigestible food to be responsible for the disturbance, then an emetic may be given, followed by a calomel purge. In very exceptional instances there may even be some justification for the employment of the gum lancet.

**Subsequent Treatment** — For a few days after a fit small doses of one of the bromides, two or three grains, should be given three daily. If the child be syphilitic or rickety then the appropriate treatment for these complaints should be vigorously employed. The greatest care should be bestowed on the diet, and on the thorough ventilation of the day and night apartments. Arrangements should be made for the child's head being raised during sleep and the feet being kept warm. If the convulsions recur with any frequency, say, every fortnight, or even every month, then a course of bromides should be prescribed. Every attack possibly lessens the resistance in the nerve tracts, so that succeeding fits occur with much slighter exciting causes than the initial ones. By allowing the fits to continue much more harm may thus be done than any that may possibly arise from the effects of the bromides on the developing tissues. It is difficult to fix a definite time, but perhaps a freedom of three months from fits should ensue before stopping the bromides. Belladonna, the salts of zinc, digitalis, ergot, musk, and other remedies that have been vaunted from time to time in the treatment of convulsions, are much less efficacious than the bromides. If the bromides fail in checking the convulsions, then borax, in two-grain doses, might be given a trial.

**Convulsive Tic.** — Involuntary muscular movements, especially of the face, accom-

pained by "explosive utterances," such as the repetition of an offensive or meaningless word, habit chorea or habit spasm. See CHOREA (*Diagnosis*)

**Cooking.** See INVALID FEEDING (*General Preparation of Food*), FOOD (*Cooking, Effects of*), PHYSIOLOGY, FOOD AND DIGESTION (*Cooking*)

**Cooper, Astley.** See HERNIA (*Femoral, Variety*), SHOULDER, DISEASES AND INJURIES (*Dislocations, Sir Astley Cooper's Method of Reduction*)

**Co-ordination.**—The harmonious action of two or more muscles or sets of muscles, under the direction of the will, and for the purpose of carrying through a complicated act. See ALCOHOLISM (*Chronic*).

**Copaiba.** See BREATH (*Clinical Diagnosis*), DRUG ERUPTIONS (*Copaiba*), NEPHRITIS (*Acute, Etiology*), PHARMACOLOGY, PRESCRIBING, PURPURA (*Symptomatic, Toxic*), URINE, PATHOLOGICAL CHANGES IN (*Odour*)—An oleo-resin obtained from various species of *Copaifera* (e.g. *C. Lamuydorfii*, *C. officinalis*, etc.), it is insoluble in water, but is dissolved in absolute alcohol, ether, benzol, etc., it contains an oil (the official *Oleum Copaiba*, given in doses of 5 to 20 m suspended in mucilage of acacia or white of egg), and a resin (containing copalvic acid,  $C_{10}H_{16}O_2$ ), it has an aromatic odour and a disagreeable taste, and it is given in doses of  $\frac{1}{2}$  to 2 fl dr (in mucilage of acacia). The chief action of copaiba is a stimulating one upon the mucous membranes of the genito-urinary tract, of the intestines, and of the lungs. It has been found to be specially useful in gonorrhoea after the acute stage has passed, it is also employed (for its disinfectant and diuretic action) in cystitis, pyelitis, and in hepatic and cardiac dropsy. Its effect upon the breath is an inconvenience, and its nasty taste requires to be disguised. See PRESCRIBING

**Copper.** See PHARMACOLOGY, PRESCRIBING, STOMACH AND DUODENUM, DISEASES OF (*General Etiology, Toxic Substances in Food*), TOXICOLOGY (*Arsenite of Copper, Copper*), TRADES, DANGEROUS (*Copper and Brass*), URINE, PATHOLOGICAL CHANGES IN (*Glucose, Fremmer's Test*)—The only official form of copper is *Cuprum Sulphas*, "bluestone." It occurs in the form of irregular deep-blue crystals, soluble in water and with a strong astringent and metallic taste. Dose— $\frac{1}{2}$ -2 gr (astringent), 5-10 gr (emetic).

Bluestone is applied to ulcers to reduce exuberant granulations, to syphilitic sores, to ulcers of the mouth, to small cancerous growths, etc. Lotions of strengths varying from 1 to 10 gr to the ounce are employed for antiseptic and astringent purposes in diseases of the eye, vagina, urethra, etc. Internally in small doses

it is given in the form of a pill in diarrhoea and bleeding from the stomach. In larger doses it is a prompt emetic, and has been used thus in laryngitis and bronchitis in children, and in cases of poisoning. It is specially indicated in poisoning from phosphorus, with which it forms a stable, insoluble compound. If it fails to produce emesis the stomach must be at once emptied by other means, on account of the powerful irritant effect of the drug. Copper sulphate has been given empirically in various chronic nervous diseases, but probably no benefit is derived from it in such cases.

**Copra- or Copro-**—In compound words *copra-* or *copro-* (from Gr *kōpos*, faeces) signifies relating to the faeces or to defecation; thus *coprocuria* is involuntary defecation, *copremia* is the depraved state of the body due to prolonged constipation and absorption of constituents of the faeces, *coproagogue* is a cathartic, *copolalia* is the use of dirty words, *copolitis* is an intestinal contraction (faecal), *coprophagy* is the eating of faecal matter, and *coprostasis* is the blocking of the bowel with a hardened mass of faeces.

**Cor-**—The heart, as used in such expressions as *Cor adiposum*, a heart showing excess of the subpericardial fat, *Cor hominum*, a hypertrophic state of the heart in which it comes to resemble a bullock's heart, *Cor bifurcatum* (see HEART, CONGENITAL MALFORMATIONS OF), a heart in which both septa are defective, and so the heart is double-chambered or reptilian, and *Cor villorum* or *Cor lusitum*, a heart covered externally with threads of fibrin, giving it a shaggy appearance.

**Coraco-**—In compound words *coraco-* (from Gr *κόραξ*, a crow) means relating to the coracoid process of the scapula, e.g. *coraco-clavicular*, *coraco-humeral*, etc.

**Coracoid Process.** See SHOULDER, DISEASES AND INJURIES (*Fracture of the Coracoid Process*)

**Coral Calculi.**—Dendritic calculi, forming in the pelvis of the kidney and constituting exact moulds of it.

**Cord.** See SCROTUM AND TESTICLE, DISEASES OF (*Spermatic Cord, Anatomy, Torsion, Haematoma, and Hydratide*), SPINAL CORD, MEDICAL, LABOUR, ACCIDENTAL COMPLICATIONS (*Abnormalities of the Umbilical Cord*)

**Cordentery.**—A teratological state met with in the chick, it is allied to omphalocephaly, and in it the notochord is elongated in the direction of the alimentary canal and in part clothed by it.

**Core-**—In compound words *core-* signifies the pupil of the eye (from Gr *kōpn*, pupil), as in *corectans*, dilatation of the pupil, *corectopia* displacement of the pupil (see IRIS AND CILIARY

**BODIES** (*Congenital Abnormalities of Iris*), *corcometer*, an instrument for measuring the diameter of the pupil, *coreoplasty*, the formation of an artificial pupil, *corestenomus*, a (congenital) narrowing of the pupil, *corocleisis*, obliteration of the pupil, *corodistichus*, dilatation of the pupil, and *coromorphosis*, the making of an artificial pupil

**Coriandri Fructus.**—The fruit of an umbelliferous plant (*Coriandrum sativum*), it contains an official volatile oil (*Oleum Coriandri*), which contains coriandrol ( $C_{15}H_{17}OH$ ) and pinene, and is given in doses of  $\frac{1}{2}$  to 3 m, coriander fruit and its oil have the carminative and stomachic actions of the volatile oils, and are used in the making of the *Confection* and the *Syrup of Senna*, in *Syrupus Rhei*, in *Tinctura Rhei Composita*, etc

**Corium.** See SKIN, ANATOMY AND PHYSIOLOGY (*Corium*).

**Corn.** See CLAVUS, TUBER DORSALIS (*Perforating Ulcer of the Foot*)—(Corns are horny indurations or thickenings of the cuticle, situated usually over one or other of the joints of the toes, and caused by pressure irregularly distributed (e.g. tight or ill-fitting boots), a corn often develops a central core (or "eye") which passes more deeply into the underlying structures and requires to be taken out before cure can be effected, treatment consists in removing the exciting cause, e.g. by the wearing of well-fitting boots, or by so distributing the pressure (e.g. by a plaster) as to prevent its concentration at any one point, the core can be taken out by soaking in hot water and then scraping, of course, if a corn become inflamed and an abscess form under it, it will be necessary to poultice and open it

## Cornea.

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See also BRAIN, CEREBELLUM (*Tumour, Anæsthesia of Cornea*), EYE, CLINICAL EXAMINATION OF (*Cornea*), GLAUCOMA (*Symptoms*), HERPES (*Ocular Complications*), HYSTERIA (*Ocular Anæsthesia*), LEPROSY (*Ulceration of Cornea*), MENINGITIS, TUBERCULOUS (*Eye Symptoms*), REFRACTION (*Astigmatism*), SCLEROTIC, DISEASES OF (*Scleritis*), SMALLPOX (*Corneal Ulcer*), SYPHILIS

THE cornea may be considered broadly as consisting of three layers the *anterior epithelium*, the *proper corneal tissue*, limited by two structureless elastic lamellæ, Bowman's membrane in front, and Descemet's membrane behind, and the *endothelium*. These parts have a common origin with the conjunctiva, with the sclerotic, and with the uveal structures of the eye respectively, and they are subject to the same changes and show the same tendencies as the parts with which they have origin. The cornea is not vascular, it gets nourishment by a system of lymph channels, and is abundantly supplied with nerves.

**EXAMINATION OF THE CORNEA.**—The cornea should be generally inspected as to shape, size, clearness, and the presence or absence of opacities. Its surface should then be examined by placing the patient opposite a window, and observing the image reflected from the various parts of the cornea as the eye follows the finger. A breach of surface can be detected by a disturbance of the image, it may then be verified by staining with fluorescein. Fluorescein 2 per cent, bicarbonate of soda 3 per cent in distilled water. The condition of the cornea generally can be best made out by examination by focal light, finally, the ophthalmoscope may be used with a lens of high power + 20 D behind the mirror.

Examination by focal light is carried out as follows.—A darkened room, a source of light, and a lens of about three inches focal length are necessary. The light is placed at about eighteen inches on one side of the patient's head, and is focused by the lens on the different parts of the cornea, the more obliquely the light falls on the eye the more clearly will opacities be seen. To obtain full advantage of this method the cornea should be magnified by a lens of high power held in the other hand.

**INFLAMMATION OF THE CORNEA—Keratitis—**

Inflammation of the cornea may be limited to the epithelial layer, to the substance of the cornea, or to the endothelium on its posterior surface, it may be a local infective process leading to ulceration or abscess, or it may be part of a disease affecting mainly the deeper regions of the eye, it is often the manifestation of a constitutional affection as in inherited syphilis. Inflammation of the cornea always gives rise to a loss of transparency, if only the anterior epithelium be involved, the surface loses its polish, and the appearance of staphling produced by its cells when highly magnified is exaggerated, sometimes the change in the cells is so marked that the magnified surface has the appearance of a piece of orange skin or shagreen. An exudation into the corneal tissue proper is shown by a yellow or gray opacity, suppuration is preceded by haziness, and the area of suppuration is surrounded by a similar area of haziness. The effect of inflammation is to produce a blurring of the parts seen through the cornea, thus the pupil is no longer quite black, and the details of the structure of the iris are obscured.

Inflammation begins with an infiltration, this consists in the appearance of numbers of leucocytes in the tissue of the cornea, the affected area is rendered opaque and the surface over it loses its polish. This exudation sometimes becomes completely absorbed, and the cornea is left quite transparent, at other times where the disturbance of the corneal tissue has been greater, or where the exudation has been present for a long time, complete restoration of transparency does not take place, the exudation is converted into fibrous tissue, which differs from the proper tissue of the cornea in not being transparent. There is a tendency, however, for old exudations to go on slowly clearing for years, as is seen in the remarkable way in which the changes in the corneal substance left by interstitial keratitis disappear. This clearance depends probably on nutrition, as we commonly see the periphery of the cornea clearing more readily and more completely than the central parts which are farthest removed from the sources of nourishment. This also probably explains the reason underlying the use of irritating remedies and massage of the cornea in the promotion of clearing of opacities of old standing.

**ULCERATIVE INFLAMMATION**—If the inflammation be of an ulcerative nature the exudation goes on increasing in amount, the corneal tissue is broken down, and either an ulcer or an abscess is produced. Suppuration generally starts in the superficial layers of the cornea, the epithelium becomes cast off from the infiltrated area and an ulcer is formed, in the earlier stages, while the margins or base of the cornea are infiltrated with leucocytes, the ulcer is *progressive*. As recovery takes place the area

of infiltration disappears, the ulcer becomes clear and transparent, but the surface is still rough. Gradually the epithelium from the edges grows over the surface of the ulcer, and blood-vessels run in from the margin of the cornea beneath the epithelium, the ulcer is healed, but its level may be lower than that of the rest of the cornea. By the development of fibrous tissue beneath the newly formed epithelium the level is gradually raised, but the transparency of the cornea is impaired by this growth of fibrous tissue, and the seat of a former ulcer may be traced for years by the opaque scar lying in the clear cornea.

Most of the pus-producing organisms have been found in ulcerative affections of the cornea. In the early days of bacteriological work on the cornea the staphylococcus and streptococcus were thought to be the most frequent provocative agents, whether the ulceration was primary or the result of injury, but owing to the work of Ulthoff, Axenfeld, Morax, and others, we now know that they are present much less frequently than other organisms. The ulcerations which occur during the course of attacks of conjunctivitis are not due to secondary infection by staphylo- or streptococci, but are caused by inoculation of the cornea with the organisms of the conjunctivitis, and we may therefore find the gonococcus, the Weeks' bacillus, the diplobacillus, and others. The characters of the ulcerative process vary with the nature of the infecting organism, and although we are not yet in the position of being able to make a new classification of corneal ulcers on this basis, yet certain facts are well ascertained. According to the researches of Ulthoff and Axenfeld, in as many as 98 per cent of the cases of acute serpygious ulcer pneumococci were present. In certain serpygious ulcers running a much less acute course, attended by little or no pain, a diplobacillus resembling somewhat the diplobacillus of chronic conjunctivitis, but having some distinguishing characters, has been found (Pétil). In bilateral infantile ulceration of the cornea (kerato-malacia) the pneumococcus has been found and also the streptococcus. The *Aspergillus fumigatus* has been found growing on the cornea in some rare instances.

The *histology of corneal suppuration* has been very thoroughly investigated by Leber, who by his observations on the cornea has added largely to our knowledge of the inflammatory process in general.

According to his observations the entrance of any pyogenic organism into the tissue of the cornea sets up hyperemia of the parts round the cornea. This is brought about by the irritative action of the toxins developed in the corneal tissue by the growth of the organism. Very soon an invasion of white cells spreads in from the periphery of the cornea, each cell



making its way through the lymph channels till it reaches the infected area. Here two things may happen: the cells either come to a standstill, killed by the intensity of the poison, and form a ring of pus surrounding the focus of infection, or where the poison is less intense, the whole of the infected region is invaded by the white-celled exudation.

The irritative action of the toxins spreads beyond the cornea to the iris and ciliary body, these become hypoeimic, the blood-vessels of the iris enlarge, the iris tissue becomes swollen, its fibres blunted, posterior synechiae may form, and from the surface of the iris and angle of the anterior chamber an exudation is poured out which sinks to the bottom of the chamber and forms a hypopyon. This hypopyon consists of a fibrinous exudation with leucocytes, and is generally fluid, so that its upper limit is horizontal, and its position varies with that of the head. A hypopyon is sterile except when the anterior chamber has been opened, when it may become infected from without. The opinion was formerly held that the hypopyon was derived directly from the seat of ulceration in the cornea by the passage of the products of inflammation through Descemet's membrane into the anterior chamber, the active resistance of this membrane and of its endothelium render this manner of access to the anterior chamber impossible. The hypopyon may sometimes be seen extending up to the region of the floor of the ulcer as a yellow cord communicating at its lower end with the general mass of the hypopyon. At the first view this would appear proof of a communication between the base of the ulcer and the chamber, but examination of the process microscopically shows that the corneal layers near the ulcer become swollen, the endothelium shares in this swelling, some of its cells exfoliate, and a deposit of coagulated lymph and cells forms on the denuded surface, which by increase comes to communicate with the general body of the hypopyon. This condition is sometimes described as posterior abscess of the cornea, it very frequently ends in a perforation of Descemet's membrane, but in the earlier stages the cells of the posterior abscess are sterile and frequently show their origin from the uvea in containing pigment.

**Results of Ulcerative Keratitis.**—If the seat of the ulcer be in the substance of the cornea itself, and the proper corneal tissue be destroyed, a permanent opacity results, the new scar tissue cannot acquire the peculiar transparency of proper corneal tissue, although the scars and blemishes left by ulcers in early life may to a great extent disappear. Such opacities are called *nebulae*, *maculae*, or *leucomata* according to their density.

If the loss of corneal substance is considerable the intraocular pressure may cause the floor of

the ulcer to bulge. At first Descemet's membrane being very elastic and resistant does not give way, but forms a hernia at the seat of ulceration, the appearance of such a hernia is that of a transparent bead on the cornea. The protrusion may flatten and become consolidated, or it may perforate, perforation is usually accompanied by a reduction of pain and irritation in the eye, but its results may be serious. The iris may come into contact with the back of the ulcer, or it may be prolapsed through the perforation. If the ulcer then heal, an adhesion of the iris to the back of the cornea will be left varying in thickness from a few filaments to a broad band of iris substance, *anterior synechia*. If after perforation the lens come into contact with the back of the inflamed cornea, an opacity is formed at the anterior pole of the lens, and an anterior polar or pyramidal cataract results. The cornea sometimes heals with much loss of substance, being then too weak to resist the pressure of fluids within the eye it bulges irregularly and forms an anterior staphyloma, this also occurs after total destruction of the cornea, the place of the cornea being taken by a thin layer of fibrous tissue united with the tissue of the iris. This weakened membrane may burst and collapse, and become permanently flattened and consolidated, or it may yield again, and again burst. At the time of its distension it protrudes between the lids like a button, and is a source of much distress, after collapsing it again ceases to give trouble for a time. After a large perforation it is not uncommon for the lens to escape, for the vitreous to become prolapsed, and for the globe to suppurate or to undergo slow shrinking.

**Causes.**—Infection of the cornea by micro-organisms is necessary for the production of the ulcerative process, in most cases this is preceded by a loss of surface epithelium. This may be the result of an injury such as an abrasion of the surface or the entrance of a foreign body, thus it is common among men exposed to injury from their trade, but it also occurs as the result of a wound, as by a baby's finger-nail, some of the worst cases are among agricultural labourers, those engaged in hedge trimming, or especially among those at work in the harvest-field. In a considerable number of cases mucocoele or chronic inflammation of the lachrymal sac is present, and thus with its decomposing contents offers a constant danger of infection to an unsound cornea. Ulcerative keratitis may also occur from infection of the small ulcers during an attack of phlyctenular disease, or in a herpetic eruption on the cornea. The cornea may become infected during gonorrhoeal conjunctivitis, when the whole of the epithelium is sodden and diminished in vitality, also where the nutrition of the cornea as a whole, and especially of its epithelium, has been profoundly altered, as in kerato-malacia. Finally, ulceration of the cornea may take place

as the result of a blood infection, as is sometimes seen during an attack of smallpox, when pustules not infrequently develop on the cornea itself.

Paralysis of the facial nerve, leading to weakening of the orbicularis muscle and to insufficient protection to the cornea, corneal anesthesia depending on fifth-nerve paralysis, the insensibility of the cornea in absolute glaucoma, and in the late stage of exhausting diseases such as cholera, may lead to a loss of surface from exposure, or from unconsciousness of the presence of a foreign body.

**Treatment**—Before beginning treatment the cause of the ulcerative process should as far as possible be sought for. Foreign bodies should be removed from the cornea and the conjunctival sac, misdirected eyelashes should be pulled out, and the condition of the lachrymal puncta should be noted. If the corneal affection is secondary to disease of the conjunctiva, treatment of the latter should be actively pursued, in gonorrhoeal conjunctivitis the nitrate of silver treatment should be carried out as long as the condition of the discharge renders it necessary, care being taken to prevent injury to the cornea by the applications or by the necessary versions of the lids. A mucocoele or lachrymal abscess should be sought for, as this probably more than any cause tends to keep up activity in an ulcer, the lachrymal sac should be opened, and its contents washed out daily, the passage into the nose should be made free, and if necessary a style should be inserted in the nasal duct.

In most cases of ulcer of the cornea great benefit will be derived from the firm application of a compress to the eye, it greatly prevents movement of the lid and the constant winking and spasm which are so painful, and it ensures warmth and protection, the eye should be first covered with a pad of cotton-wool and the bandage should be applied over it. Where there is much conjunctival secretion, as in the cases of abscesses secondary to purulent conjunctivitis, it is best not to bandage the eye, because of the danger to the cornea of keeping the infectious discharge pent up between the lids.

Heat applied to the eye is a valuable means of treatment, especially when the general level of nutrition is low. The most convenient method of applying heat is by hot bathing, simple hot water, or better, hot saline, containing 1 per cent of chloride of sodium, or a solution of 2 per cent of boric acid, should be applied to the eyes by pads dipped in the solutions kept as hot as they can be borne, this bathing should be kept up for half an hour at a time, and should be frequently repeated. A good way of keeping the lotion hot is to insert the basin in which it is contained in a basin of very hot water which can be frequently renewed. A means of applying heat that is very valuable is a Japanese hand-warmer, a small oblong tin

box containing a slowly burning cartridge, if this be applied to the eye by a bandage over a layer of cotton wool, the heat can be kept up without difficulty for any length of time. Caution must be mentioned here against the use of any lotion containing lead where there is loss of the corneal epithelium, an insoluble, densely opaque film of lead salts may be deposited on the ulcerated surface.

*Atropine* should be used so that the pupil may be kept fully dilated, iritis is present in many cases of corneal ulceration, and atropine is necessary, to prevent adhesions and to subdue the iritis, apart from this, however, atropine secures physiological rest of the eye by dilating the pupil and paralyzing the ciliary muscle. The best means of using atropine is in the form of an ointment of the strength of four grains of the alkaloid to the ounce of vaseline, a small piece should be inserted inside the lower lid twice a day, or more often if iritis is present. The ointment is preferable to drops, as it remains longer in the conjunctival sac, and acts more thoroughly on the eye, with less risk of the unpleasant symptoms of atropine poisoning from its absorption into the system. Atropine should be used just sufficiently to maintain full dilatation of the pupil, it should not be smeared freely over the lids, sometimes where its use has been too profuse or too long-continued it gives rise to a highly congested state of the skin and conjunctiva, a condition known as atropine irritation. Atropine irritation occurs generally in those who are peculiarly susceptible to its action, in such people the smallest amount gives rise to symptoms. Where much redness of the skin and conjunctival irritation comes on during the use of atropine this form of irritation should be borne in mind, otherwise it may be thought that the ulcer is worse and needs more atropine. The drug should be discontinued and some other mydriatic, such as scopolamine or duboisine, should be substituted for it if necessary, but it is better to discontinue all mydriatics and use a simple ointment of boric acid for the irritated skin. It is sometimes useful to combine iodoform with the atropine in infective ulcers. Atropine 4 gr., iodoform 5j, vaseline, 3j.

*Eserine* has been strongly recommended by some surgeons as a routine treatment for ulcers of the cornea. Where there is a possibility of perforation it has undoubtedly a valuable place, but, speaking generally, its use tends considerably to the increase of irritative symptoms, pain, and intolerance of light, and to the circumferential congestion and iritis. It should be reserved for those cases in which imminent danger of a perforation of the cornea leads to the fear that an extensive prolapse of the iris may occur. Eserine is also of value in certain cases in which the ulcer is indolent for a long time, neither advancing nor healing—a condition depending, probably, on impairment of nutrition. The increase of

vascularity in the neighbouring parts induced by eserine promotes the nutrition of the cornea, the ingrowth of blood-vessels, and the healing process in the ulcer. It may be used as an ointment, or as drops in the strength of one or two grains to the ounce.

Where the ulcer is progressive, with an advancing infiltrated edge, these measures are insufficient, recourse must be had to the *actual cautery* or to *strong caustic applications* to the seat of the ulcer. The cautery is most conveniently applied by a flattened loop of platinum wire bent to a convenient angle, and raised to a dull-red heat by a galvanic current. The ulcer being stained with fluorescein all the parts which take on the stain should be burnt, especially where the infiltration is densest. It is not necessary to burn the whole surface of the ulcer, the part that is healing and covered with endothelium will not stain, and should not be touched.

If the galvanic cautery be not available, a small Paquelin's or a metal cautery heated in a spirit-lamp may be used, but these are much less handy. If the temperature of the wire be not greater than a dull-red heat the cautery does not produce effects beyond the parts actually touched by it, if the thin base of the ulcer be perforated the incandescent wire is at once chilled by the escaping aqueous and no harm results.

For most advancing ulcers treatment by strong local applications is enough, the ulcer should be stained, the infiltrated parts should be scraped with a small sharp spoon, and a camel-hair brush dipped in the fluid should be painted over the stained surface, and introduced into all the pockets and recesses of the ulcer. Of these caustic applications pure carbolic acid is one of the most efficient and least painful, where it touches the cornea the tissue is at once whitened and afterwards cast off, so that it should not be applied carelessly or in excess. Other agents, such as nitrate of silver, 10 grains to the ounce, perchloride of mercury 1 or 2 per cent, or tincture of iodine, may be used, but the pain from them as a rule lasts longer and is more severe than after carbolic acid.

Before using any of these methods of treatment the eye should be thoroughly cocaroused and a speculum introduced, as immobility of the eye is very essential, a small amount of solid cocaine in fine powder may be applied to the ulcer and also to the conjunctiva where it is gripped by the fixation forceps. After catenarsation atropine ointment or atropine ointment with iodoform should be used, and a compress. Section of the base of the ulcer, opening the anterior chamber, a method originated by Saemisch, is a very valuable treatment where a hypopyon is present, or when there is risk of perforation. A Graefe's knife is passed into the anterior chamber at one side of the ulcer, across the chamber to the other side, the cutting edge being directed forwards, the knife is made to

cut its way out, completely dividing the base of the ulcer, opening the anterior chamber, and as a rule causing the escape of the hypopyon. This should be done slowly to avoid the sudden escape of the aqueous, and with it prolapse of the iris and possible damage to the lens.

It is sometimes advisable to keep the wound open for a time in the event of the reappearance of the hypopyon, this may be done by inserting a small probe between the lips of the wound daily.

A recently prolapsed iris may be replaced, but it is generally safer to remove it, it should be seized, freed from the edge of the ulcer all round, drawn slightly forward, and cut off at the level of the cornea. The cut ends will then either retract or may be replaced, but it is seldom possible to avoid adhesions of the iris to the wound.

*Subconjunctival injections of various antiseptic fluids* have been strongly recommended by some surgeons for the treatment of corneal affections, and especially of the different forms of infective ulcer, perchloride and cyanide of mercury have been used most frequently. More recently, however, it has been found that sterilised valine solution, 0.75 per cent of chloride of sodium, injected beneath the conjunctiva is equally useful and less irritating. Enough is injected to produce a moderate distension of the loose conjunctiva all round the cornea, there is a certain amount of reaction after the injection for about twenty-four hours.

This form of treatment has not found much favour in this country, and is now less spoken of even by those who first used it.

In dealing with perforations of the cornea in which the iris is entangled, there is frequently a difficulty in obtaining a firm cicatrix, the involvement of the iris leaving unsound spongy tissue. In some cases a fistula is formed, in others the cornea over the seat of the perforation remains thin and bulged for an indefinite time, the risk of septic inoculation of such an eye is considerable. Complete rest in bed, avoidance of movement and use of the eyes, a compress over the damaged eye, the other one being also bandaged, are means by which sounder union may be secured. Eserine has been recommended to keep down the tension of the eye as much as possible, but there is little or no evidence that eserine reduces the tension except where it has been previously raised.

A method of considerable value in securing firm cicatrization is the *transplantation of conjunctiva to the seat of the ulcer*.

After freshening the surface of the damaged cornea or the edges of the fistula, this may be done in the following way—A flap of conjunctiva is dissected up, being left attached by a pedicle, the flap is twisted on itself and tucked into the weakened spot, another method is to dissect up a bridge of conjunctiva at the upper

part of the globe, leaving the two ends attached, bringing the bridge down to the seat of the ulcer, and tethering it by one or two sutures applied to the conjunctiva at the edge of the cornea; it may also be done by dissecting up the conjunctiva all round the globe, and bringing it together by continuous suture at its free edge, thus covering the whole cornea.

The conjunctiva seems to act in two ways, partly by the support which it gives to the cornea, and partly by becoming adherent to the seat of the ulcer. The fact of the cornea being covered by conjunctiva does not seem to give trouble, after the sutures come away the conjunctiva falls back again to its proper place.

Oxygen gas has been recommended as a treatment for indolent ulcers of the cornea, the application is made by means of a closely fitting cap, the oxygen being passed over the eye in a constant stream.

#### *Treatment of the Results of Corneal Ulceration*

**Opacities**—Little can be done for the treatment of opacities of the cornea. The use of stimulants, like the yellow oxide of mercury ointment, 4 gr to the ounce of vaseline, or of wine of opium, combined with massage, should be continued for a long time. Opacities due to a deposit of lead on the cornea may be removed by scraping. In some cases undoubted improvement has followed the application of a galvanic current directly to the cornea, but it is unreliable. Tattooing is sometimes done, partly for cosmetic reasons and partly for the improvement of vision. When the nebula partly covers the pupil, or where an artificial pupil has been made opposite clear cornea, the dispersion of the light passing through the nebula causes much disturbance of vision through the clear part of the cornea. Rendering the nebula dark by tattooing it, may result in great improvement of vision. The surface of the nebula is pricked all over by a bundle of needles, and Indian ink rubbed up into a paste is applied to the surface, or a Wecker's grooved needle, containing Indian ink in the groove, is passed into the cornea parallel to its surface, and the ink deposited in the substance of the cornea, the operation has to be repeated several times. Too much should not be done at one sitting, eyes in which the cicatrised cornea is thin, or has the iris adherent to it, should not be touched. The pigmentation of the cornea so obtained is not permanent.

Transplantation of the cornea is not yet within the domain of practical surgery, for though the operation has been done with success as regards the life of the transplanted cornea, its transparency has never been maintained.

#### *Types of Corneal Ulceration—The Simple Ulcer.*

The simple ulcer may arise from an injury to the epithelium, or from the introduction of a foreign body into the eye. It appears as a somewhat raised grey spot, which

becomes a shallow crater with some infiltration of its base, there is circumcorneal congestion, pain, and intolerance of light.

**Treatment**—Hot fomentations, compress, and atropine (see general remarks on Treatment).

**Infective Ulcer**—The ulcer heals rapidly as a rule, but it may occasionally penetrate deeply, and even perforate the anterior chamber. In this case, instead of clearing, the base and margins of the ulcer become yellow with infiltration, the surrounding cornea becomes hazy, iris is set up, and a hypopyon forms.

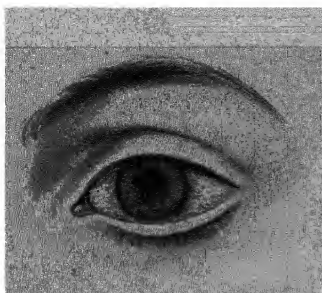
This type of infective ulcer is most commonly seen during the course of a purulent conjunctivitis, but it may occur as the result of an infection of the corneal tissue apart from general conjunctivitis. Its tendency is to penetrate deeply into the cornea, but not to spread widely, in this respect it differs from the serpiginous ulcer.

**Treatment**—On the same lines as that of the simple ulcer, but more active measures, such as carefully applied caustics, or the cautery, may be necessary. If purulent conjunctivitis be present its active treatment should be pursued.

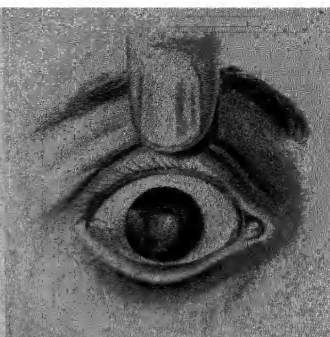
**Circumferential Ulcer**—During an attack of conjunctival catarrh we sometimes meet with one or two rounded or crescentic ulcers near the margin of the cornea. They usually heal readily. During the later stages of trachoma, when pannus of the cornea has been formed and the lid has cicatrised, it is not uncommon to see small sharply-cut ulcers with a clear base at the edge of, or on the pannus itself, they generally come with a slight recurrence of irritation in the lids. In addition to the local treatment of atropine the lids themselves require attention.

**Serpiginous Ulcers**—There are several kinds of ulcer whose tendency is to spread into the healthy parts in their neighbourhood, in their whole course the character of these ulcers is distinct, the only common feature is their tendency to spread. The main forms are—the acute serpiginous ulcer, the marginal ditch ulcer, the rodent ulcer, and the dendritic ulcer. Some of these are described now, the others will be found in their place later.

**The Acute Serpiginous Ulcer, Ulcus Serpens, Hypopyon Keratitis**—This begins as a dirty grey spot showing a loss of surface, with an infiltrated base and edges, the base is uneven, and is covered with broken-down epithelial and corneal cells. It commonly follows a slight injury, and occurs especially in those who have a diseased lachrymal sac. The characteristic feature of this form of ulcer is its advancing edge, which generally appears as a yellow crescentic area situated at one edge of the ulcer. This crescentic area tends gradually to spread over the cornea superficially in one direction, while the part of the ulcer first formed



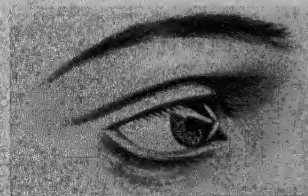
Cornea—Keratitis punctata.



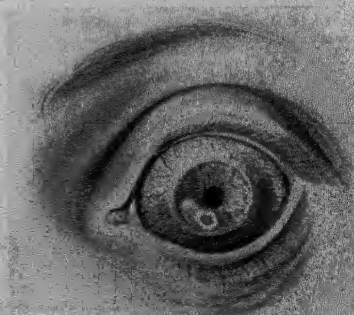
Cornea—Interstitial keratitis.



Buphthalmus.



Conical Cornea.



Ulcer of Cornea.



remains stationary or tends to heal. The cornea around the ulcer is cloudy, there is intense circumcorneal injection, a certain amount of iritis is present, the iris is blurred and swollen, enlarged blood-vessels may be seen on it, and posterior synechiae may be formed. A hypopyon appears very early in the disease. The signs of irritation are very great in most cases, intense pain, intolerance of light, some fever, and a want of sleep, in other cases the signs of irritation are not marked. If not checked the hypopyon increases and may fill the anterior chamber, the ulcer spreads so that a considerable area of the cornea becomes involved, and this either yields to the intraocular pressure and perforates, or the ulcer penetrates deeply and the same result obtains. In some cases perforation is followed by general infection, panophthalmitis, and total destruction of the eye, but, on the other hand, it sometimes marks the onset of a remission of most of the irritative signs, and is followed by a tendency towards healing. Abscess of the cornea is closely allied to the serpigulous ulcer, but it spreads in the substance of the cornea without destroying the surface, if it is seated in the superficial layers the greater part of its anterior wall will break down and form an ulcer, but it may invade the deeper layers and run its whole course without destroying the surface. It is generally not very acute, but it is frequently accompanied by iritis and hypopyon, in some of the very chronic cases the mass of exudation does not go on to active suppuration, but quietly subsides, and is eventually transformed into fibrous tissue.

**Treatment**—Fomentations and atropine in the earliest stages, with a compress, followed by the galvanic cautery or the local application of carbolic acid, or some other caustic, Saemisch's operation on the ulcer, the treatment of any lachrymal sac complication (see general remarks on Treatment).

**The Marginal Ditch Ulcer** *Marginal Ring Ulcer*—This begins as a groove near the edge of the cornea, it is usually rather deep, with sharply-cut edges, with or without infiltration of its base, its tendency is to creep gradually round the edge of the cornea, but it may heal in one part while advancing in another, if not checked the whole cornea may be destroyed by it. It generally occurs in old people with failing nutrition, the cause is not known.

**Treatment**—The galvanic cautery should be applied to the advancing edge of the ulcer. Esmerine should be used to improve the local nutrition as well as heat and compresses.

**The Rodent Ulcer**—A grey infiltration appears at the periphery of the cornea and soon breaks down into an ulcer, the ulcer does not penetrate deeply, but tends to spread slowly and intermittently over the whole corneal surface. The appearance of this form of ulcer is quite

peculiar and characteristic. The surface of the ulcerated area is below the level of the rest of the cornea, it is uneven and opaque, and may contain blood-vessels which have spread in from the limbus. At the line of junction with the unaffected part of the cornea, the latter stands up considerably above the ulcerated area, and has abrupt cliff-like edges, which are grey in colour and undermined in places. The progress of the ulcer is very slow, and it has periods of quiescence, but it eventually creeps over and spoils the whole surface of the cornea. It occurs chiefly among elderly people, and especially among those who have lived abroad, but it is a very uncommon form of ulcer. The only treatment at all efficacious is to cauterise the advancing grey edge of the ulcer.

**Kerato-Mycosis Aspergillus**—This rare affection has been described by several Continental writers, but no case has hitherto been reported in this country. It is due to the growth on the cornea of the fungus *Aspergillus fumigatus*, which gains an entrance by means of a foreign body or other injury to the corneal epithelium. The fungus appears as a grey mass with a dry crumbling surface, seated generally near the centre of the cornea, the signs of irritation are not severe, and the course of the affection is tedious. The mass of the growth is, as a rule, cast off by exfoliation of the part of the cornea in which it has been growing. Its removal may be hastened by scraping.

**THE PHLYCTENULAR ULCER** (*Phlyctenular conjunctivitis*, *Postular conjunctivitis*, *Erythematous or eczematous conjunctivitis*, *Phlyctenular or postular keratitis*, *Marginal keratitis*, *Hesper of the cornea*, *Serosofolous or strumous ophthalmia*, *Lymphatic conjunctivitis*).

This disease appears in a great variety of forms, each of which has different stages, nearly all the forms and stages have received separate names. The result has been the unnecessary complication of a very common affection. The disease is mainly one of childhood, the general type consists in the formation of a limited elevation of lymphoid cells beneath the epithelium of the cornea or conjunctiva, the epithelium breaks down, leaving a small shallow ulcer. The ulcer remains for a time, but is gradually repaired by epithelium creeping over its surface, at the same time a similar ulcer has been formed elsewhere. Sometimes the ulcers are very minute, and are known as milium phlyctenules. These are often so numerous as to invade the whole limbus, and even to be scattered over the whole surface of the cornea, which has the appearance of being covered with minute grains of sand. The irritative symptoms are severe, the conjunctiva of the lid is much swollen, there is intense fear of the light and spasm of the lids, the outer canthus is frequently cracked or excoriated, the child shrinks away to the darkest corner and keeps his face deeply

buried. At the slightest attempt to open the eyes a copious gush of tears comes out, and it is well-nigh impossible to obtain a view of the cornea. These confluent phlyctenulae produce a swelling of the limbus, while the cornea in the immediate neighbourhood loses its transparency, and contains a number of small grey points just beneath the epithelium. With small phlyctenulae, as a rule resolution takes place, the elevations disappear. If they break down into an ulcer by loss of the covering epithelium, the ulcer as a rule heals, but it may become infected and take on the characters of an infective ulcer (see p. 232). In other cases the behaviour is quite different: the ulcer assumes a chronic course and may remain stationary for weeks, vessels grow into the cornea from the limbus towards the crater-like depression, and healing takes place slowly. The healing does not always follow the ingrowth of vessels, the ulcer slowly pushes its way towards the centre of the cornea, carrying the leash of blood-vessels with it. This condition is known as a *Leish ulcer* or *Fuscular keratitis*. The advancing edge of the ulcer is convex towards the centre of the cornea, and somewhat raised in the form of a crescent, consisting of an exudation into the corneal substance, the vessels of the fasciculus ending in the concavity of the crescent. The difference between this leish ulcer and the natural healing of an ulcer by an ingrowth of vessels lies in the fact that the leash ulcer contains a yellow edge of purulent exudation. This condition may last weeks or months, and results in the formation of a hard-like opacity which remains visible for years.

Where the ulcers of the cornea are multiple and adjacent to one another the ingrowth of vessels of repair from the limbus may occupy a considerable area of the cornea. These vessels remain as potential blood-channels for a very long time after the complete healing of the ulcer, and are liable to become easily congested, giving rise to a condition known as phlyctenular pannus, or superficial vascular keratitis. The vessels lie beneath the epithelium, at times they are invisible except to careful examination, and at others they are much enlarged, and the corneal surface is rough and uneven.

*Caution*.—This is a disease of childhood, but it does not occur generally in children under one year old, its most common period is from two to sixteen years, during this time skin eruptions, eczema, and impetigo about the face are very common, together with a similar condition of the nasal mucous membrane. It most commonly occurs when the changes in temperature are great and sudden, as in spring and autumn, as the effect of climatic changes is greater among people who live in damp, ill-ventilated, or overcrowded houses, so this disease is common among the poor, and rarely occurs in children whose surroundings are satis-

factory. As a probable result also of environment the children fall into irregular habits of feeding, and eat anything at any odd time instead of having regular meals, whereby the appetite becomes capricious and the desire for unwholesome food is established.

The next most common exciting cause is a previous attack of some acute exanthematous disease, such as measles, scarlet fever, or whooping-cough, by far the most common of which is measles. The eye affection comes on a few weeks after the measles, and is not the same as the conjunctival inflammation which commonly ushers in the first stage of measles. The irritation of head lice is a frequent accompaniment of the disease, and is especially likely to occur, as during this period the children appear sufficiently morose and unattractive to make neglect probable.

It is held by many that this affection is in its nature stromous or scrofulous. The old term *Scrofulous ophthalmia*, besides including all the eye affections of this group, was applied to all cases in which there was extreme intolerance of light, it was thought that the photophobia was induced by an irritation of the optic nerve and retina, and a consequent reflex spasm of the orbiculars, but inasmuch as treatment applied to the terminations of the fifth nerve in the cornea and conjunctiva leads to diminution of the intolerance of light, we know that the affection is a superficial one only, the intolerance is not greater than may be produced by a foreign body under the upper lid. It is characteristic of all cases of superficial keratitis in the growing period of life that irritation, intolerance of light, and spasm of the lids are much greater than in adult life. Yet the term *stromous* or *scrofulous ophthalmia* has this justification, that in a large number of cases of superficial corneal inflammation in the young there is a certain condition of the tissues which makes them favourably disposed to the occurrence of inflammatory outbreaks which are very chronic in their course, and which tend to recur without obvious cause.

This condition is associated with a proneness of the lymphatic tissue in the body to hypertrophy, the glands in the neck or at the angle of the jaw become enlarged, the mucous membrane of the nose becomes swollen and inflamed, and masses of adenoid tissue form in the pharynx. Also if the family history and antecedents of the parents be carefully examined there will be found a liability to tuberculous affections in others of the family. Evidence of this sort has to be received with caution, as it depends on the statements of friends who are often anxious to produce what they think the inquirer is seeking to find. But the liability to recurrence of phlyctenular disease, its association with other lymphatic hypertrophies, the frequency of its appearance in more than one



member of a family, point to a peculiarity in the tissues capable of being transmitted from one generation to another, and favourable to the growth of certain organisms. The actual relationship with tubercle is probably no nearer than this phlyctenule have been excised and examined frequently without revealing the presence of anything but staphylococci, tubercle bacilli have never been found.

**Treatment**—This should be local and general. Locally a little boric acid lotion, ten grains to the ounce, should be dropped into the eye about three times a day, if there is any photophobia or spasm of the lids atropine should be used night and morning. It is necessary to get a view of the cornea—often a matter of difficulty, this can generally be done by some coaxing, it may be necessary, however, to use lid retractors, or even to give an anæsthetic to get a view. In many of the cases in which the spasm of the lid is greatest the involvement of the cornea is only trivial. It is often possible to obtain relief of the spasm by the sudden shock of cold water falling on the face from a sponge held above the patient, or if the spasm is great and the corneal involvement small, it is useful to evert the lids, dry the conjunctiva, and apply to it freely and to the skin of the lid, especially if eczema be present on the carthus to be cracked, a solution of nitrate of silver, mopping up the excess, a few hours after the application the child sometimes spontaneously opens the eyes. When the stage of irritation has passed, yellow oxide of mercury, four grains to vaseline one ounce, may be used for a considerable time.

The fascicular form is very tedious, if the crescentic area is yellow and advancing it must be cauterised, or scraped and touched with carbolic acid, or with the point of a stick of integrated nitrate of silver.

As a rule it is better not to bandage the eyes, coolness, free access of air are good, to protect the eyes from light, goggles or a shade may be used, bleeding and blisters are useless and harmful, a seton in the scalp is a very active remedy in cases which resist all other kinds of treatment, but it is liable to set up cellulitis of the scalp with suppuration in the neighbouring glands, so that it should only be used in those cases in which all other methods have failed.

As to *general treatment*, it is well to begin with a purge followed by iron, arsenic, or quinine and cod-liver oil or cream as an addition to the diet, regulation of the hours of feeding, the forbidding of cakes or mummy fruit between meals, a change of air to the country or seaside is most beneficial, if this cannot be secured, the child should be made to live in the open air as much as possible. The local skin affections should be treated, adenoids removed, and disinfection of the nose and throat secured.

**AFFECTIONS OF THE CORNEA ASSOCIATED WITH THE FORMATION OF BITES. II.—Herpes of the Cornea**

—This affection appears under two forms, simple febrile herpes and the more severe herpes zoster.

Febrile herpes occurs chiefly in children during a febrile attack, such as exanthematous fever, pneumonia, or even a catarrh of the respiratory or nasal passages. During the early stage there are intense photophobia, spasm of the lid, and lachrymation, one or more minute blisters form on the surface of the cornea, and when seen some of them may be entire, containing fluid, or more generally they have burst, and we see a shred of epithelium attached to the edge of a shallow pit in the cornea. The bullæ may be arranged in line, or they may form a constellation in one part of the cornea, the edge of the pit is quite clear and sharp, and appears as if a piece of the cornea had been cut out by a spud or finger-nail. The healing generally takes place as in an ordinary ulcer after the shedding of the epithelium. The sensibility of the cornea is said to be lowered in the region of the blisters, and the tension is also said to be reduced, it is difficult to apply satisfactory tests in the condition of irritation present, but the tension is not always lowered, and the cornea is sometimes hyperæsthetic, these may vary in the different stages of the affection. Febrile herpes is only characteristic in the earliest stage, when it is not often seen, at a later period it cannot be distinguished from a simple or phlyctenular ulcer.

**Treatment**—Rest, shade for both eyes, atropine, and general treatment of the catarrhal state are measures generally enough to heal the ulcer without leaving a mark, but it may become infected like any other ulcer (see general remarks on Treatment of Ulcers of the Cornea).

*Herpes zoster of the cornea* occurs as part of herpes zoster ophthalmicus, an inflammation of the first division of the fifth nerve or Gasserian ganglion. Where the supra-orbital or supra-trochlear branches only are affected the cornea as a rule escapes, but when the nasal branch is involved, shown by the spots occurring on the tip of the nose, there are generally ulceration of the cornea, iris, and sometimes cyclitis, the nerves of the front of the eye are derived from the nasal branch of the fifth nerve by way of the long root of the lenticular ganglion. The herpetic attack is preceded by intense neuralgia, pain and swelling of the lid, this latter is so great that the affection is often thought to be erysipelas, one or two vesicles appear on the cornea and form ulcers, in spite of the intense pain the cornea is as a rule completely anæsthetic, and may remain so for a long period.

The *treatment* of the ulceration is that of a simple ulcer, but it must be borne in mind that the cornea is insensitive and cannot protect itself against foreign bodies and other kinds of injury, so that it must be bandaged and carefully watched.

The *dendritic ulcer* is a well-defined variety of superficial corneal ulceration. It is generally seen as a groove in the cornea consisting of a central stem with small buds or branches at each side, the ulcer has a tendency to creep over the cornea and spoil a large part of the surface. The ulcer sometimes occupies the centre of the cornea, but in the most characteristic cases extends from the limbus as a broad trunk which gives off branches on each side, the branches themselves ending in secondary branches or buds. In its earliest formation the affection is herpetic in character, the buds have the appearance of small bullae. The appearance of the dendritic ulcer is very striking and beautiful after staining with fluorescein.

It is not known what is the cause or exact nature of the dendritic ulcer, but it probably belongs to the herpetic group, it occurs frequently in people who have been subject to malaria, and has been described as *malarial keratitis*, but in this country it is seen not seldom in patients who have never been abroad and who have never had any form of malaria. It occurs in those who are past the best period of life, or who are temporarily run down, I have seen it twice in young children, both of whom were much exhausted by prolonged joint suppuration.

Treatment must be radical. The course of the ulcer is rarely stopped by the ordinary routine measures, such as fomentations, atropine, compresses. The galvanocautery at a very low temperature may be used, but there is risk of producing a permanent scar with it, carbolic acid is safer and better, but several applications are often needed. The most effective treatment is absolute alcohol applied by dipping a small roll of lint into the alcohol and rubbing the ulcerated surface with it, the epithelium for some distance around the ulcer is removed, but it is quickly regenerated. The only drawback to this very efficient method, which we owe to Swanzy, is its painfulness.

An affection closely allied to febrile herpes is the *superficial punctate keratitis* (Fuchs). A number of small grey elevations appear in the cornea arranged like febrile herpes in lines or constellations, their number may vary from ten or twenty to about a hundred, grey lines are also seen in the cornea, which are probably caused by exudation into the corneal nerve trunks. There is much photophobia, although the cornea is somewhat anæsthetic. The attack is part of a general catarrhal condition affecting the parts supplied by the fifth nerve, the irritation clears up in a short time, but the grey spots may remain for months.

*Local Treatment*—Protection from light, and atropine (see general remarks on Treatment).

*Recurrent Bullous Keratitis*—A solitary blister sometimes forms spontaneously under the corneal epithelium, attended by severe

neuralgia, intolerance of light, and watering. Sometimes there has been an injury at the spot, and for a time recurrences take place in the same eye, but the affection is often independent of injury, and the relapses occur either at definite times in the year or at intervals depending on some variation in health, in some cases the attack always occurs at a menstrual period, though not at each one.

*Treatment*—Bandage, rest, atropine, sometimes tearing off the shred of epithelium left by the bulla, causes considerable relief of pain. Quinine has been advised as a prophylactic, on the assumption of a malarial basis, but it is not possible to predict the onset of an attack.

Bulla are sometimes formed in eyes blind from old glaucoma, in which the cornea is more or less devoid of sensation. They also occur upon old leucomata associated with a temporary increase of tension, it may be necessary to do an iridectomy for the cure of the increase of tension if the eye is worth saving.

*Filamentary keratitis* is a very rare disease. Numerous small threads are seen hanging from the surface of the cornea and attached to it quite firmly. The threads consist of a twisted strand of epithelial cells having a bulbous end, and covered in places by mucus. When the threads drop off the base heals, but their place is taken by others caused by degeneration of adjacent epithelium. The treatment consists in rest, atropine, and bandage.

*Kerato-Malacia*—*Infantile Ulceration of the Cornea*—This serious affection occurs in young children who are the subjects of grave disturbance of nutrition, due either to starvation or improper feeding, or to the exhaustion of disease. The first manifestation of the affection is a dryness of the conjunctiva and cornea, and the formation of a small triangular foam patch at the inner and outer margins of the cornea in the exposed part of the eye. This lack of lustre or dryness, which varies greatly in degree, is due to a fatty degeneration of the epithelial cells, and a consequent inability of the tears to moisten the surface.

Local infection of the cornea through the degenerated epithelium is followed by deep infiltration and rapid destruction of the whole or part of it, the actual organisms present have been found to be streptococci or pneumococci, and the foam patches contain the virulent bacillus.

During its whole course the disease is attended by very slight local symptoms, little or no discharge, and scarcely any intolerance of light. Kerato-malacia is rarely present without there being grave disturbances of general nutrition, the children, if not actually wasted, have an unhealthy, ashen colour, they are, however, for the most part greatly emaciated by constant diarrhoea and vomiting, and in spite of every care they die.

Kerato-malacia appears in England mostly in hand-reared infants badly nourished in consequence of unsuitable food or of prolonged diarrhoea. Poorness or insufficiency of milk, the use of condensed milk from which the cream has been separated, the use of patent starch foods in the place of pure milk, are among the most common causes, but it may occur in hand-reared infants in the feeding of whom the nutritive value of the milk has been impaired by prolonged or repeated boiling being carried out with the laudable desire of rendering it sterile. One is reminded of the experiment of Majendie, who fed a well-nourished dog on nothing but pure white sugar and water, with the result of producing a central ulcer of the cornea. Severe exhaustion after acute fevers or pneumonia is a predisposing cause, and is often seen in the late stages of mild ophthalmia neonatorum in babies suffering from congenital syphilis. In countries where religious fasting is strictly observed it also occurs in breast-fed children.

*Treatment*, both local and general, should be directed towards improvement in nutrition. Warmth by compresses and hot fomentations frequently applied, the local use of esuine in the form of an ointment, one grain to the ounce three times a day, are the best local measures, while general treatment should be in the direction of securing proper food, milk diluted according to age, cream or cod-liver oil, and meat juice, and the diarrhoea should be combated.

*ULCERATION OF THE CORNEA FROM EXPOSURE*.—When the cornea is exposed to the air it becomes rapidly dry, and if not moistened by tears its surface is destroyed and a way provided for the entrance of organisms.

This condition is produced by cicatrization and contraction of the lids from old injury or disease, it occurs in facial paralysis when the lower lid no longer keeps in position, also in the extreme protrusion of the eye sometimes met with in exophthalmic goitre, and in orbital tumours. It is also seen during the condition of apathy in patients suffering from exhausting disease.

*Treatment*.—The eye should be closed temporarily by a piece of strapping, but if the exposure is likely to be permanent, the edges of the lids may be pared and sutured together for a certain distance.

*NEURO-PARALYTIC KERATITIS*.—When the fifth nerve is paralysed the cornea becomes anæsthetic and is readily exposed to injury, foreign bodies are not swept away, and the surface is no longer kept moist by the movements of the lids induced by the sensations of the cornea. The epithelium becomes cast off from a portion of the surface, and an ulcer is formed, this form of ulceration runs a very chronic course, and is little affected by treatment, infection may take place, and the eye may be lost. The changes have been

described as beginning interstitially without loss of epithelium.

It is held by many that the paralysis of the fifth nerve is associated with a trophic disturbance in the cornea, and although the results of section of the fifth nerve on the nutrition of the cornea tend to show that if the cornea be protected such section has no influence on it, yet if we take the analogy of other parts, e.g. the glossy skin which occurs in paralysis of sensory nerves, it is highly probable that the fifth nerve does exert an influence on the nutrition of the cornea. (Imreal evidence is in favour of this view.)

The *treatment* is that of ulcerative keratitis, especial care being taken to secure protection of the eye.

*INTERSTITIAL OR PAINFUL KERATITIS*.—In this disease the deeper layers of the cornea are attacked by chronic inflammation with the formation of opacities and with the development of blood-vessels. The changes in the cornea show a tendency to clear up for months after the inflammatory symptoms have subsided, there is no tendency to ulceration or suppuration.

The appearance of the changes in the cornea is preceded by circumcorneal congestion, lachrymation, and intolerance of light, an examination of the eye is made difficult by spasm of the lids, hence in many of the worst cases it is not possible to say what is the exact condition of the cornea. The changes show themselves in many forms, but the most common mode of onset is the appearance at the periphery of the cornea in one spot of a group of macule deep in the corneal substance, the epithelium covering them becomes swollen and loses its lustre, so that the macule are only seen dimly. At the same time the vessels of the limbus become much enlarged at this spot, and the limbus itself is swollen and encroaches somewhat on the surface of the cornea in the form of a vascular crescent, this encroachment does not exceed certain narrow limits. At the same time, there is an ingrowth into the corneal substance of vessels coming from the deeper layer of the sclerotic, appearing beneath the limbus and having no connection with its vessels. These are long and thin, and are in the substance of the cornea, they have the appearance of long tufts, and have been likened to the fibres of a beson, they gradually force their way into the substance of the cornea, the area of macule still extending in front of them towards the centre, sometimes the process starts from two opposite sides of the cornea, the skinning line of macule advancing towards the centre till they meet those coming from the other side. In another group of cases, instead of starting peripherally from one or two points, the macule make their appearance towards the centre of the cornea, where they become con-

fluent, and from this region gradually extend towards the margin, the cornea is almost uniformly grey, the centre being more opaquely grey than the periphery. At the same time the epithelium is swollen and loses its polish, with a magnifying glass or corneal microscope the individual cells can be seen unevenly ocellated. There are varieties in the amount of vascularity present, in some cases the vessels can be seen singly, in others they are very numerous and appear only as a pink patch in the cornea, a condition known as a *salmon patch*. The density of the opacity varies greatly, and is, as a rule, patchy, at the height of the attack the deeper parts of the eye cannot be seen with any clearness, but, as a rule, it can be made out that the anterior chamber is deep, that the pupil is either not dilated freely or that adhesions are present, and that there is a deposit on the back of the cornea, *keratitis punctata*, the sign of involvement of the ciliary body. The tension of the eye is generally rather lowered. In older patients from the beginning the corneal opacity in its earliest stage sometimes takes on the striated form, grey lines are seen deep in the corneal substance, these are followed by the development of a general haze of the deepest layers of the cornea.

Interstitial keratitis is always chronic in its course, the opacity of the cornea and the inflammatory and irritative symptoms go on increasing for about two months, and then slowly decline. At its worst the vision is much reduced, even to the perception of hand movements. As the attack abates the ciliary congestion diminishes, the cornea clears from the periphery, and the sight begins to improve, the clearing and improvement in vision continuing for many months. The severity of the attacks varies very greatly in young children it is not uncommon to see a few macule at the periphery, or a slight central haze with ciliary congestion disappearing entirely in a very few weeks, an attack in immature only, on the other hand, in the worst cases the irritative symptoms may last twelve months or longer, and the cornea may be converted into a dense white tendinous structure absolutely opaque. It happens rarely that the cornea yields owing to inflammatory softening and becomes bulged, this is sometimes uniform, and may subside without causing any gross change in the corneal curvature, at other times the yielding of the cornea may be preceded by the formation of a gummata of the cornea, a local mass of granulation tissue surrounded by a dense felt of vessels, and having a tendency to degenerate at the centre. It is very uncommon for an abscess or ulcer to form, the gummata, like most of the manifestations of interstitial keratitis, undergoes slow involution in most instances.

The participation of the uveal tract of the eye in interstitial keratitis is one of the marked

features of the disease. It is difficult to form an estimate of the depths in the cornea of the changes even with a highly magnifying lens especially as in the period of evolution the irritative symptoms prevent careful examination, but the deposits in Descemet's membrane are generally well-marked dots of varying size, sometimes densely crowded and very fine, at other times large, like spots of grease, these are either scattered generally over the back of the cornea, or chiefly collected at the lower part. Occasionally the appearances suggest an almost universal change in the endothelium in certain areas of the lower part of the cornea, as if there were an irregular deposit of amorphous ciliary granules on it.

Iritis is shown by hyperæmia of the iris, posterior synechie, and exudations into the pupillary area, the failure of the pupil to dilate under atropine is sometimes marked, even when on recovery there can be found little or no sign of past iritis, the failure of dilation probably arises from the absence of absorption of the atropine through the inflamed cornea. After recovery it is often possible to make out recent extensive peripheral choroiditis, either in the affected eye or its fellow. The share taken by the uveal tract varies from a degree in which it can scarcely be verified to almost pure iridocyclitis, with slight involvement of the cornea only.

A rule, with scarcely any exception, is that the disease occurs in both eyes, the interval between the two, however, may be as much as from three to four years, recurrences may take place, but after a typical well-developed attack they are rare. Among young children it is not uncommon to see slight attacks of interstitial keratitis or hybrid interstitial and phlyctenular attacks recurring several times, followed at a somewhat later period in life by a well-developed attack of interstitial keratitis, after which there is no more trouble.

It is well to warn the patient's friends, as soon as the nature of the affection is recognised, of its probable duration, of the almost certain involvement sooner or later of the other eye, and also of the ultimate probable recovery of very useful vision. Nothing is so encouraging to the patient as the assurance of recovery during the long weeks of suffering.

*Causes of Interstitial Keratitis*.—It is more common in females than in males, the age of greatest prevalence is from about seven to eighteen, but these limits are not very strict. I have seen a few cases in quite young children under the age of two, and it is not uncommonly seen up to twenty-five, and sometimes up to thirty years.

The most frequent cause of interstitial keratitis is hereditary syphilis, which in most of the cases may be recognised by its stigmata without having to question the parents directly. But

evidence of repeated miscarriages, or of premature births, or of great mortality among the children in early life, may be easily obtained, as also the testimony as to infantile skin eruptions or snuffles.

The signs by which hereditary syphilis may be recognised are by the shape of the cranium, the physiognomy, the teeth, and the choroid.

The frontal eminences are prominent and rounded, the central part of the face is depressed owing to the sunken bridge of the nose and to the flattening of the front of the superior maxillary bones. The mouth itself shows linear cicatrices radiating outwards especially from the angles, and frequently depressed cicatrices are seen in the skin of the neighbourhood. Shotty lymphatic glands may be felt in the neck, and the throat shows signs of old ulceration. An examination of the choroid of the other eye by the ophthalmoscope will often reveal the scars of old disseminated choroiditis.

The appearance of the teeth is one of the most characteristic signs of inherited syphilis: in the milk teeth there is nothing characteristic, but the incisors very often become carious. In the permanent teeth attention should be directed to the upper central incisors, although the others may also show signs. In the upper central incisors two main types are met with, those in which the cutting-edge is narrowed and has a central notch in it, and those in which the notch is not present, but the cutting-edge is much narrower and thinner than the crown of the tooth, the screw-driver type. The teeth are also undersized and separated from one another by spaces.

Other manifestations of inherited syphilis are deafness from internal ear disease, periosteal nodes, or effusions into the knee-joints.

Interstitial keratitis also occurs rarely in acquired syphilis, its course is much the same as in the hereditary disease.

Cases occur occasionally in which the most searching examination of the patient fails to reveal any of the signs of hereditary syphilis, and in which no evidence in favour of it can be obtained from the family history. Such patients are frequently in bad health and are rapidly losing flesh, it is not possible to demonstrate the presence of tubercle in such cases, but the probability of it is great, especially where there are nodular growths springing from the angle of the anterior chamber, as sometimes occurs in such cases. Microscopically nodules very suggestive of tubercle have been found in the angle of the anterior chamber, and the tubercle bacillus has been identified in the cornea in one case by Zimmermann.

The opportunities for examining by the microscope an eye in the acute stage of interstitial keratitis have been very rare. Such an examination shows a cellular infiltration of the posterior half of the cornea increasing in density as the

posterior elastic lamina is approached, at this point the cellular infiltration is so great that the cornea appears to consist entirely of round-celled exudation, its proper structure is hidden. Descemet's membrane is thrown into folds, and there are deposits of round cells upon the endothelium. Newly-formed blood-vessels are present in the deeper layers of the cornea. The anterior layers appear normal. The round-celled infiltration also invades the ligamentum pectinatum, the iris, and ciliary body.

*Treatment*—Much may be done to alleviate pain and distress in interstitial keratitis. Protection from light should be secured by dark goggles, or a shade made to cover both eyes. Heat, by means of hot compresses or fomentations, is occasionally very useful in reducing the discomfort of the patient. Locally we should carefully avoid the use of any kind of irritant as long as the disease is advancing. Atropine, if given to the ounce of vaseline, should be used to prevent or limit the effects of iritis, and its use should be maintained until the height of the disease is well past. When the symptoms have begun to abate, irritant or stimulant remedies may be begun, the chief among which are the yellow oxide of mercury ointment, which should be begun cautiously in a strength of four grains to the ounce. Calomel may be flicked into the eye, or hot steam applied, or wine of opium dropped in. If there is any return of irritation then use must be discontinued. The cornea may be further stimulated to clear by massage combined with the use of the yellow ointment. This treatment may be continued for months or years.

As a rule most cases do not require bandaging, but if there is a probability of the cornea yielding to pressure it must be supported.

In the *constitutional treatment* mercury should be used, the method by which it is given perhaps the most satisfactory for prolonged use, or mercury with chalk may be given, care being taken that the patient be not salivated. As the patients are often anemic and much depressed, this treatment should be combined with syrup of the iodide of iron and cod-liver oil, at the same time general hygienic measures, warm clothing, good food, and fresh air should be secured. In any case the results of treatment are not brilliant, the disease appears to run its course unchanged and to invade the second eye while the patient is still under treatment for the first. However, there is something to be done by constitutional treatment, from observation of cases at the Victoria Hospital for Children during a series of years. I found that the cases which were treated by mercury ran a shorter course and had less tendency to recur than those not so treated, and also that the complications, such as extreme deafness, were more frequent in those not under mercurial treatment.

*LOCALISED INTERSTITIAL KERATITIS OF ADULTS*

(Keratitis profunda).—A grey opacity appears in the deeper layers of the cornea near its centre, it consists of macule or striae, sometimes the centre of the opaque area is comparatively clear, the opacity forming a not very dense ring round the centre, the striae are generally seen in the earlier stages, and when the opacity is fully developed they disappear. There is frequently deposit on the back of the cornea, and evidence of iritis or irido-cyclitis, and vessels may grow into the cornea from the periphery. At its height the condition of the eye much resembles the interstitial keratitis of hereditary syphilis. There is generally not a great amount of irritation, the attack lasts a month or six weeks, and passes off, leaving a certain amount of permanent opacity. The patients are generally beyond middle life, and only one eye is as a rule attacked.

The causes of the disease are obscure. It has been ascribed to cold, to rheumatism, to malaria, many of its subjects suffer from defective terminal circulation, cold hands and feet, dyspeptic troubles, and constipation. The local treatment should be protective glasses or a bandage, hot fomentations and atropine, 4 gr. to the ounce of vaseline, the general treatment should be in accordance with the requirements of each case.

**SIRIATED KERATITIS**.—The appearance of grey lines in the corneal substance occurs in a variety of conditions, delicate grey lines are often seen extending at right angles to a wound in the corneal substance, whether the whole cornea has been cut through or not, they are sometimes present extending from the area of a deep ulcer, they occur in some cases of iritis and irido-cyclitis, and are not infrequently the earliest form taken by the opacity in interstitial keratitis, they are sometimes double-contoured, suggesting a tube containing an opalescent fluid. They are said to be sometimes caused by a compress which has been worn for a long time. Their most common appearance is after cataract extraction, extending from the wound at the upper periphery of the cornea downwards into the substance of the cornea, they are more common where the cornea has been much bruised during the operation, but a certain amount of striation is present after nearly every extraction.

Striated keratitis is at most a temporary phenomenon, it either disappears entirely after a short time, or gives way to some more permanent general haziness. The explanation of the occurrence of the lines is not simple, they probably have more than one cause, there is no structure in the normal cornea which corresponds with them, but certain tubes can be made to appear in the cornea by injecting between its lamellae mercury or air under pressure, Bowman's tubes. Some of the opaque lines are to be explained by the natural formation of Bowman's tubes, by the pressure of fluids in inflammatory conditions of the parts adjacent

to the cornea. They have been formed experimentally by making sections of the cornea in rabbits, and have been then found to be caused by wrinkling of Descemet's membrane, the kind which follows cataract extraction may have this cause.

Leprosy sometimes attacks the cornea, nodules form generally at the margin of the cornea, associated with iritis, they generally break down, leading to loss of the eye, the cornea is also sometimes anæsthetic.

**SCLERONING KERATITIS**.—A relapsing, persistent, subacute inflammation of the ciliary region involving the adjacent part of the cornea, and sometimes attended by iritis. The recurrences generally occur in fresh places, and the general effect of each attack is to leave conical or triangular areas of opacity at the margin of the cornea with the base directed outwards, these areas are sometimes very dense and blue-white in colour, and approximate in appearance to the sclerotic.

The cause is unknown. The local treatment should be rest, fomentations, and atropine.

**Calcareous Film of the Cornea (Transverse Calcareous Band)**.—This is a form of degeneration which occurs in the superficial layers of the cornea, in the shape of a broad band or oval patch of dirty grey colour lying in the part of the cornea that is commonly exposed. It lies just beneath the epithelium, and consists mainly of fine calcareous granules united with hyaline substance. Its development is very slow, it starts on each side of the cornea, and gradually progresses towards the centre.

It occurs mostly in eyes that have been lost from old irido-cyclitis, but is occasionally met with in old people whose eyes are otherwise healthy.

**Treatment**.—If the eye is otherwise good the film may be removed by scraping and afterwards treating the cornea as if it had been injured.

**Congenital Opacities of the Cornea**.—In one variety of congenital opacity the cornea appears to have the structure of sclerotic, the opaque sclera seeming to extend for a certain distance into the cornea, and only gradually thinning into clear cornea. Such eyes are often microphthalmic, the condition may be seen in more than one member of the family.

In other cases a fine ring of opacity is seen just inside the corneal margin, it has the same appearance and situation as the arcus senilis (arcus juvenilis).

Other opacities are due to fetal inflammation, such as interstitial opacities of various kinds. In congenital hydrophthalmos the whole cornea is sometimes faintly milky.

The *arcus senilis* is a non-inflammatory degeneration of the cornea, appearing as a grey line at the upper and lower parts, and sometimes meeting at the outer and inner, it is densest above and below, and is separated from

the sclera by a thin band of transparent cornea. The grey line is due to a hyaline change in the tissues of the cornea, and has no significance.

*Blood-staining of the cornea* is found when the anterior chamber has been filled with blood for a long time. The cornea appears to be of a red-brown colour, and is quite opaque, the absorption goes on very slowly from the periphery. The staining is due to deposit in the cornea of blood crystals which have formed after diffusion of the blood colouring matter through Descemet's membrane.

*Keratitis Punctata*—This term has already been used, it is commonly applied to the deposits on the back of the cornea that are met with in inflammations of the ciliary body. A number of fine dots is seen on the back of the cornea, sometimes evenly scattered, but more often found on a triangular area at the lower part of the cornea where they are formed by deposition. The dots consist of shreds of fibrin and leucocytes cast off from the ciliary body and deposited on the corneal endothelium. Where they are not soon removed by absorption they produce destructive changes in the endothelium. Some of these dots are of large size, and have the appearance of spots of grease on the back of the cornea. It is possible that these deposits may increase in size, when examined in the fresh state they have been found to contain colonies of bacteria (Snellen).

*Conical Cornea*. The central part of the cornea begins to bulge very gradually without inflammatory symptoms, forming a blunt cone, in the more advanced cases the conicity is readily seen by looking at the profile of the cornea. The disease makes itself felt by a disturbance of vision, objects can only be seen clearly when held close to the eye, yet the sight is scarcely improved by concave glasses. The slighter degrees may be recognised by the ophthalmoscope by a shadow seen on the background of the eye, crescentic or annular in shape, and shifting with the movements of the mirror, the condition may also be recognised by examining the corneal reflex with a Placido's disc, and comparing it with that from a normal cornea.

The disease may come to an end spontaneously, or in the worst cases a nebula may be formed at the apex of the cone which is rather below the centre of the cornea, perforation never occurs.

The affection is a rare one, it attacks both eyes in young adults, especially women. It is atrophic in nature, and is due either to some developmental peculiarity in the corneal tissue, or to defective nutrition in the part farthest removed from the blood-vessels.

*Treatment*—In the early stages some improvement may be effected by prescribing concave glasses with cylinders, the proper strength of which may be found out by the shadow test, these may be used either alone or combined with

an opaque screen having a small hole or narrow slit cut in it. Vision has been improved in many cases by hyperbolic glasses as suggested by Rahlmann.

Of other methods of treatment eserine or pilocarpine used continuously has been said to reduce the conicity, but in most cases it is of no use.

Operative measures, with the object of flattening the cone by forming a resistant cicatrix at or near it, are to be recommended where the impairment of vision is very great.

The apex of the cone may be removed by shaving it off with a Graefe's knife without opening the anterior chamber, and after two days touching the raw surface with mitigated nitrate of silver stick. An elliptical flap may be removed from the apex of the cone, and the cornea afterwards sutured, a small disc of the outer layers of the cone may be separated by a Bowman's trephine, and the surface allowed to heal and contract. Multiple punctures may be made into the apex of the cone with a fine needle, and repeated after some weeks, or the galvanocautery may be used at a very dull heat to burn the surface layers of the cornea, if perforation take place during burning no harm will result to the deeper parts if care be used, as the cautery is at once chilled by the escaping aqueous. This last method has the advantage of being free from the risk of infection.

*Hydrophthalmos* (Buphthalmus).—The cornea undergoes general and progressive enlargement, and sometimes becomes hazy in this affection. The enlargement is part of a general enlargement of the globe, and may be looked upon as congenital or infantile glaucoma.

*Tumours of the Cornea*.—Tumours of the cornea are rare. Myxomata, fibromata, sarcomata have been described as well as epitheliomata starting from the limbus, and cysts following injuries, formed by the inclusion of cells from other parts in the corneal substance.

*Injuries*.—*Foreign Bodies in the Cornea*.—Foreign bodies, fragments of steel or iron, small pieces of stone or particles of ash from locomotive engines, imbedded in the cornea, are very common accidents, and give rise to varying amounts of pain and irritability.

They sometimes remain for long periods without exciting disturbance, as among those engaged in metal grinding, in whom the cornea may often be seen studded with minute fragments of stone which have been there an indefinite time. Scales of seeds or the wing-cases of small insects may adhere to the cornea by their concave surfaces.

Particles of steel or iron become partly oxidised, and the foreign body may be often seen surrounded by a brown ring, which consists of sodden epithelium impregnated with oxide of iron. This ring may remain behind after removal of the particle, and continue to keep the

eye irritable. If not removed, foreign bodies are generally cast off by exfoliation of the part in which they are lying, during the whole of this time the eye is liable to infection at the seat of the injury, especially where the lachrymal sac is not healthy.

For their removal the eye should be thoroughly cocainised, two drops of a 2 per cent solution of cocaine should be put into the eye three times at intervals of a minute, the patient should be seated facing a light, the surgeon standing behind him. The lids being held open by the left index and middle fingers, the foreign body may be removed, at first by the edge of a piece of clean blotting-paper, or if it is imbedded by a spud or needle. The ring of oxide round the foreign body should also be removed. If the anterior chamber be penetrated other measures must be taken to prevent the object from being pushed into the chamber, or the parts beneath from being injured. If of iron or steel, the attempt should be made to remove it with a strong magnet, this is often unsuccessful owing to complete oxidation of the metal.

Eserine should then be used, one or two drops of a solution of 2 gr to the ounce, to contract the pupil and protect the parts beneath from injury. A keratome or broad needle should be passed into the anterior chamber and held against the back of the cornea behind the perforated spot by an assistant. This will prevent the foreign body being thrust into the chamber by the attempts at removal from the front of the cornea which must now be continued.

After removal of any foreign body in which the corneal surface has been broken, a compress should be kept on till the wound is healed, and atropine should be used as for the treatment of a corneal ulcer, but where the anterior chamber has been opened the atropine should not be used until it has become closed.

Sometimes the track of a perforation made by a particle of metal can be seen in the cornea without any trace of the object itself. The iris should be examined carefully, the pupil should then be dilated, the lens minutely inspected, and the fundus systematically examined by the ophthalmoscope. Finally, in cases of doubt or difficulty the X-rays may be used to determine the presence or absence of a foreign body.

Wounds of the cornea made by blunt or jagged instruments are often irregular, and their edges will not come into apposition. If the iris is protruding, the exposed part should be drawn slightly forward free from the edges and cut off, the edges of the cut iris should then be tucked back. It is not advisable to attempt to return to the interior of the eye a piece of iris which has been bruised in the wound and exposed to septic contamination. A very useful measure in closing gaping wounds of the cornea is to dissect up a flap from the adjoining conjunctiva and place it over the wound in the cornea

after thoroughly washing the parts with an irrigator.

**Burns.**—After scalds by hot water, or burns by acids or alkalis, the cornea looks steamy and dull. The extent of the injury depends to some degree upon the nature of the agent effecting it, alkalis, lime, or sulphuric acid produce serious and permanent damage, whereas even strong nitric acid or blistering fluid may effect only temporary damage. In the worst cases the cornea appears dry and white, and is completely anæsthetic, this condition is likely to end in complete necrosis. Lime burns are among the most common injuries, but fortunately the lime is nearly always partially slaked, the full effect of lime or caustic alkalis is produced very slowly, and only as recovery takes place can the amount of cicatrization be appreciated. In every injury of this kind the prognosis should at first be strictly guarded even where the change appears to be slight only, owing to cicatricial changes the conjunctiva may be drawn more or less over the cornea, or adhesions may form between the lid and the cornea.

**Treatment.**—All trace of the caustic agent should be removed, treatment should be by rest, a compress and atropine, a few drops of a solution of 2 gr to the ounce once a day. The contractile cicatrization should be prevented as far as possible by putting in pure castor oil three times a day, and once a day allowing free movement of the eye in all directions, carefully separating the lid from the eye.

**Injuries of the Cornea.**—Superficial abrasions are extremely painful, and cause much watering, hyperæmia and intolerance of light owing to the friction of the lid against the denuded surface. These injuries are frequently caused by branches or leaves of trees, or by the finger-nail. The loss of surface is generally confined to the epithelial layer, and is not easily seen unless we examine the corneal reflex.

**Treatment.**—The firm application of a compress and atropine, in the form of an ointment (4 gr to the ounce), cocaine gives temporary relief, but the prolonged use of it is likely to produce softening of the epithelium. As sleep is often impossible it may be advisable to give a hypnotic. Rest and immobility of the eye for a few hours are enough to secure regeneration of the epithelium.

Relapses of abrasion are sometimes seen without any fresh injury (see "Relapsing Bullous Keratitis").

**Cornet Player's Emphysema.**  
*See LUNGS, EMPHYSEMA (Inducing Causes)*

**Corn-flour.** *See DIER (Vegetable Foods, Cereals), INVALID FEEDING (Diet during Convalescence, Corn-flour)*

**Cornu.**—*Cornu*, literally a horn, means a horn-shaped process or projection, e.g. of a cavity,



such as that of the lateral ventricle of the brain, thus, there are the anterior and posterior grey cornua of the spinal cord, the cornua of the thyroid cartilage and body and of the thymus gland, the cornua of the lateral ventricles of the brain, the cornua of the hyoid bone, and the cornua of the uterus

**Cornutina.**—An active alkaloid of ergot  
*See* ALKALOIDS

**Corona Radiata.** *See* PHYSIOLOGY, NERVOUS SYSTEM (*Cerebrum, Corona Radiata*), GENERATION, FEMALE ORGANS OF (*Ovaries, Microscopic Appearances*)

**Coronal Suture.** *See* LABOUR, PHYSIOLOGY (*Third Factor, Passenger, Fetal Head*)

**Coronary Arteries.** *See* HEART, PHYSIOLOGY OF (*Coronary Circulation*), HEART, MYOCARDIUM, AFFECTIONS OF (*Chronic Interstitial Myocarditis, Arteriosclerosis of Coronary Arteries*), PHYSIOLOGY, CIRCULATION (*Flow of Blood through the Heart*)

**Coroner.** *See* MEDICINE, FORENSIC (*Certification of Death, Procedure in England and Wales*)

**Corpora.** *See* CORPUS

**Corpora Conduplicata.** —Birth "with body doubled up," occurring very rarely in cases of transverse presentation *See* LABOUR, DIAGNOSIS AND MECHANISM (*Transverse Lies, Spontaneous Delivery*)

**Corpulence.** *See* OBESITY

**Corpus.**—A body (plural *corpora*), a frequently employed term in anatomy, *e.g.* corpora Alauti, corpus callosum, corpora cavernosa, corpus denticulatum, corpora lutea, corpora oryzoides (rice-like bodies in joints), corpora quadrigemina, corpus striatum, corpus vitreum, etc

**Corpuscle.**—A cell, a cell-like body, or an aggregation of cells, *e.g.* red and white blood corpuscles (*see* ANEMIA, BLOOD, *Cellular Constituents*), colostrum corpuscles (*see* COLOSTRUM, MILK), corpuscles of Graafy, Krause, and Vater, Malpighian corpuscles, Laveian's corpuscles (*see* MALARIA), Pannian corpuscles, etc

**Corrigan's Button.** *See* CAUTERY (*Actual*)

**Corrigan's Pulse.** *See* HEART, MYOCARDIUM AND ENDOCARDIUM (*Physical Signs in Different Forms of Heart Disease, Pulse in Aortic Incompetence*)

**Corrigens.**—The *corrigen*s is the ingredient in a prescription which corrects the action of the *basis* or principal ingredient *See* PRESCRIBING.

**Corrosive Sublimate.** *See* MERCURY (*Hydrargyri Perchloridum*), CHOLERA, EPIDEMIC (*Diagnosis*), TOXICOLOGY (*Corrosive Poisoning, Mercurial Poisoning*)

**Corsets.** *See* PREGNANCY, MANAGEMENT, PELVIS, PERINEUM AND PELVIC FLOOR (*Prolapsus Uteri, Causes*), PUERPERIUM, PATHOLOGY (*Sore Nipples, Predisposing Causes*), SPINE, SURGICAL AFFECTIONS OF (*Spinal Curves, Treatment by Plaster Jackets*)

**Cortex.**—The outer part of the substance of the brain, the kidney, the ovary, the suprarrenal capsule, etc (*see* BRAIN, GENERATION, FEMALE ORGANS OF, etc.), also the bark or rind, *e.g.* *cortex sambuci* (sambucus bark)

**Corti, Organ of.** *See* PHYSIOLOGY, THE SENSES (*Hearing, Internal Ear*)

**Corybantism.**—"A state of excitement (probably hysterical) accompanied by choreic movements, fantastic visual hallucinations, and sleeplessness" (Hack Tuke), the name takes its origin from the corybantes or priests of Cybele, who behaved in a delirious fashion at their celebrations

**Coryza.**—Nasal catarrh *See* NOSE, ACUTE INFLAMMATION (*Acute Rhinitis*), NOSE, CHRONIC INFLAMMATION (*Coryza in Children*), NOSE, NASAL NEUROSES (*Coryza Favo-motora Periodica, Coryza Edematosa*), NOSE, ACCESSORY SINUSES, INFLAMMATION (*in Acute Coryza*), ATROPHY, INFANILE (*Diagnosis, Suppurative Coryza*), COCAINE (*Uses, Acute Coryza*), MEBALLES (*Cowse*), MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Respiratory Symptoms*)

**Coscinium.**—False calumba or the dried stem of *Coscinum fenestratum*, official in the Indian and Colonial Addendum to the British Pharmacopoeia, it has the same action and uses as ordinary calumba root (*q.v.*), and there are three preparations of it, viz the *Infusum Coscini* (dose,  $\frac{1}{2}$  to 1 fl oz), the *Liquor Coscini Concentratus* (dose,  $\frac{1}{2}$  to 1 fl dr), and the *Tinctura Coscini* (dose,  $\frac{1}{2}$  to 1 fl dr)

**Cosmetics.**—Operations or medical applications, etc, for the purpose of restoring natural beauty, such a plastic procedure as the repair of a hare-lip is a cosmetic operation, and various powders and ointments (some of which contain lead) are used for "improving" the complexion *See* DERMATITIS TRAUMATICA ET VENENATA (*Causal Agents, Chemical Compounds*), TRADE, DANGEROUS (*Lead-Poisoning*)

**Costa- or Costo-.**—*Costa-* or *costo-* in compound words means relating to a rib, thus *costalgia* is intercostal neuralgia, *costo-coracoid* is related to a rib and to the coracoid process of the scapula, etc

**Coster's Paste.**—A paste containing 120 grains of iodine dissolved in one fluid ounce of light oil of wood tar, it is sometimes employed as a parasiticide in cases of ringworm.

**Costiveness.**—Constipation, or, more correctly, a less degree of intestinal inefficiency than is met with in constipation. See CONSTIPATION.

**Cotarnina.**—An alkaloid,  $C_{12}H_{11}NO_9$ , obtained from narcotina ( $C_{22}H_{21}NO_7$ ) by oxidation, its hydrochloride is *stypticin* (*q v*). See ALKALOIDS, STYPTICIN.

**Coto Cortex.**—Coto bark is a non-official drug, used sometimes to check diarrhoea; there is a *Tractura Coto*, of which the dose is 10 in every two hours (with mucilage), the active principle is *cotoin* ( $C_{22}H_{18}O_{10}$ ), a glucoside, and has been used for the same purpose.

**Cotterill's Operation.** See BRAIN, SURGERY OF (*Wagner's Operation, Modification of*).

**Cotton Root Bark.**—*Gossypii Radicis Cortex* or cotton root bark is got from the *Gossypium herbaceum*, and is official in the Indian and Colonial Addendum to the British Pharmacopoeia, its preparations are the *Decoctum Gossypii Radicis Corticis* (dose,  $\frac{1}{2}$  to 2 fl oz) and the *Extractum Gossypii Radicis Corticis Liquidum* (dose,  $\frac{1}{2}$  to 1 fl di), it has the action and uses of ergot.

**Cotton-wool.**—*Cotton*, *Gossypium*, or *cotton-wool* is the hair of the seeds of various species of *Gossypium*, if the oil have been removed it is known as "absorbent cotton-wool", if not, as "non-absorbent wool". See also COLLOIDUM, PYROXYLINUM, TRADES, DANGEROUS (*Textile Trades, Cotton Workers*). Cotton may be impregnated with various medicinal substances (antiseptic, hæmostatic), and it is then known as iodoform cotton, salicylic cotton, iodised cotton, sublimated cotton, hæmostatic cotton (containing ferric chloride and alum), and Nankeen cotton (containing picric acid).

**Cotugno's Disease.** See SCIATICA.

**Cotyledon.** See FOLIUS AND OVUM, DEVELOPMENT OF (*Placenta et Teina*).

**Cotyloid.**—Literally, *cotyloid* (from *κότυλη*, a cup, and *εἶδος*, resemblance) means cup-like, in anatomy it signifies relating to the acetabulum. See HIP-JOINT, DISEASES.

**Couch Grass.** See AUROPYRUM.

**Couching.**—An operation employed in cases of cataract for breaking down the opaque crystalline lens (*q v*) by means of a couching-needle in order to produce reclinatio or absorption. See CATARACT (*Treatment, Operative, Reclinatio*).

**Cough.** See AORTA, THORACIC, ANEURYSM (*Symptoms*), BRONCHI, BRONCHIAL GLANDS (*Pressure Effects, Diagnosis*), BRONCHI, BRONCHITIS (*Symptoms*), BRONCHI, BRONCHIECTASIS (*Symptoms*), CHILDREN, CLINICAL EXAMINATION OF (*Respiratory System, The Cough*), GOUT (*Respiratory System*), HYPNOTISM, HYSTERIA (*Disorders of Respiratory Organs, Cough*), LARYNX, BENIGN GROWTHS OF (*Symptoms*), LIVER, DISEASES OF (*Hepatopneumonia, Symptoms*), LIVER, PERIHEPATITIS (*Signs and Symptoms*), LIVER, CONGESTION, LUNG, TUBERCULOSIS OF (*Lung Symptoms*), LUNGS, EMPHYSEMA OF (*Causes*), MEDIASTINUM (*Chronic Mediastinitis, Symptoms*), PHYSIOLOGY, RESPIRATION (*Special Respiratory Movements*), SPASM (*Spasmodic Cough or Cynocha Hebetis*), STOMACH AND DUODENUM, DISEASES OF (*General Symptomatology*); THYROID GLAND, MEDICAL (*Esophthalmic Goutre, Symptoms, Respiratory System*).—Cough is an important symptom in many diseases, and may constitute the chief complaint of the patient, while in other diseases the presence and character of a cough hardly noticed by the patient may give important aid in diagnosis.

*The Act of Coughing*.—Coughing is mainly an expiratory act in which air is expelled explosively through the mouth. Air is drawn into the chest and shut in by the closure of the glottis. The intrathoracic tension is increased. The gates of the larynx are then opened, and the imprisoned air escapes explosively. The gates of the larynx are the true and the false vocal cords. It is by the apposition of the false vocal cords that the intrathoracic tension can be increased to a sufficient degree to permit of an explosive cough. In animals, such as the sheep, in which the false cords and the ventricles of Morgagni are undeveloped, there is no proper explosion on coughing.

Cough may be voluntary, but is usually reflex. When the irritation is not too strong, the impulse to cough can be voluntarily restrained.

The impulse to cough commonly results from irritation of some of the branches of the vagus, and may proceed from—

- 1 The respiratory mucous membrane (nasopharynx, larynx, trachea, bronchi).
- 2 Aural irritation, as from foreign bodies in the external meatus, or rarely from chronic otitis.
- 3 The back of the tongue, through the lingual branch of the superior laryngeal nerve.
- 4 Gastric irritation—the "stomach cough" which occurs in association with chronic dyspepsia both in children and adults.
- 5 Irritation of branches of the vagus by enlarged bronchial glands, or by enlarged glands, tumours, or abscesses in the mediastinum.
- 6 The skin, especially of the throat and chest.

Apart from local irritation, cough may be purely "nervous."

THE CLINICAL VARIETIES OF COUGH.—*Disorders*

of the *Larynx*.—In simple and in membranous laryngitis the cough is commonly hoarse, barking, and imperfect, or, as is usually said, croupy. In ulceration or thickening of the mucous membrane of the larynx the cough may be husky and imperfect, or may be loud and clanging.

When a malignant tumour or an aneurysm presses upon the trachea, or involves the recurrent laryngeal nerve, the cough is commonly loud, resonant, and hoarse, and has been compared to the cry of a gander.

In paralysis of both vocal cords the patient is unable to cough, in abductor paralysis, whether unilateral or bilateral, the cough is unaffected, in adductor paralysis there is perfect cough combined with aphonia.

*Diseases of the Lungs* — In *bronchitis* the cough is at first short and dry, and may be painful, as secretion increases it becomes moist, and may be paroxysmal. Severe paroxysmal cough may be present in *fibroid phthisis*, and, more characteristically, in *bronchiectasis*. In *phthisis* cough is one of the early symptoms. At first it is dry and hacking, later it becomes loose, and is attended with muco-purulent or purulent expectoration. When cavities develop the cough may become paroxysmal, and is often most severe in the morning. Occasionally advanced, and even advancing, phthisis may be present with entire or almost entire absence of cough. This occurs especially in hæmic patients. In *pneumonia* the cough is frequent, short, dry, restrained, and associated with severe pain in the side. In acute *pleurisy* cough is usually, but not always, present. It is seldom so distressing as in acute pneumonia.

The paroxysmal cough of *pertussis* is very characteristic. A series of coughs follow each other so rapidly that no inspiration can occur. The patient becomes cyanosed, the veins of the forehead and scalp become distended, the eyes become prominent, and the tongue is often protruded. Bleeding from the nose may occur. At last a sudden inspiration takes place with a loud whoop. This may be followed immediately by a second or by several series of coughs and whoops before the paroxysm is over. At the end of the paroxysm a quantity of mucus is generally expelled with some violence, and in young children vomiting is usual. During the paroxysm the child seizes hold of its nurse or of some piece of furniture for support, in order to give purchase to the accessory muscles of respiration. In pertussis the cough is apt to be most troublesome at night.

Enlarged glands at the root of the lung may give rise to very severe and persistent coughing, sometimes paroxysmal in character, either by pressure on a bronchus or by irritation of branches of the vagus. When symptoms of pressure on a bronchus are absent it is often difficult to make out the cause of the cough. Eustace Smith has called attention to a physical

sign often met with in these cases. If the child is made to throw the head as far back as possible, a bruit is heard on auscultation over the manubrium sterni which disappears when the head is brought forward again. He attributes the murmur to pressure on the left innominate vein.

It must never be forgotten, that the irritation of the pulmonary branches of the vagus giving rise to cough may itself be secondary to some other condition, for example, measles, typhoid fever, or some other infectious disease, to chronic heart disease or to Bright's disease.

*Diseases of the Heart* — Pericarditis is sometimes attended by a hard painful cough. Valvular disease, and especially mitral stenosis and mitral incompetence during the stage of failing compensation, are accompanied by cough, often with watery or blood-stained expectoration.

Aneurysm of the aorta may give rise to very distressing cough, either by direct pressure on trachea or bronchus, or by irritation of the recurrent laryngeal nerve.

Any condition in which the heart's action is feeble, and where consequently the circulation through the lung is carried on imperfectly, may give rise to a troublesome cough.

*Diseases of the Abdominal Viscera* — The "stomach cough" associated with indigestion has already been mentioned. The existence of a true "stomach cough" has been doubted, and it may be admitted that such a diagnosis may legitimately be regarded with some scepticism. In many cases of the kind the cough appears to be due to pharyngeal irritation associated with the dyspepsia. The irritation produced by entozoa is said occasionally to give rise to cough. Cough may be present in pathological conditions of the ovaries and tubes, and pregnancy is also regarded as an occasional cause.

*Nervous Cough* — The term "nervous cough" may be applied to all forms of cough in which no cause of local irritation can be discovered. Nervous cough may be a mere habit, more annoying to the hearer than to the subject. Its occurrence is often associated with emotion, particularly the emotion of speaking in public. Some forms of nervous cough have already been referred to.

*Hysterical cough* is comparatively common in cases of hysteria. It may be very severe and persistent, and give rise to serious apprehensions as to the condition of the lungs.

The *periodical night cough* occurs in children. The cough comes on in paroxysms about the middle of the night, and may last for several hours. It is probably often due to irritation of the vagus by enlarged lymph nodes.

About the time of *puberty* a loud barking cough, recurring at frequent intervals, is not uncommon in boys of neurotic constitution.

*Diagnosis* — Some forms of cough, such as the cough of pertussis or of aneurysm, are very

characteristic, and many more are easily traced to their origin. The difficulty in diagnosis is greatest where no obvious disease of the pharynx, larynx, trachea, bronchi, or lungs can be made out, nor of the heart or aorta. Such cases should not be too quickly set down as nervous. Repeated examination should be made of all possible sites of local irritation. In children the ears and the naso-pharynx should be carefully examined, and the possibility of enlargement of glands of the tracheo-bronchial group must be borne in mind. The family history should also be inquired into, especially with reference to any tendency to tuberculosis or neuritis. Hysterical cough may be present in either children or adults. In women the possibility of pregnancy or of pelvic disease being responsible must be considered. In adults, and especially in elderly patients, any evidence of feebleness of the circulation, such as weakness of the pulse or coldness of the extremities, is of importance.

**Treatment.**—The treatment of cough due to diseases of the respiratory organs or of the heart and aorta will be found detailed under the appropriate headings. Excluding these, the throat will frequently be found to be the seat of irritation, and pharyngitis, enlarged tonsils, or elongated uvula will require local treatment. Gargles, sprays, pigments, pastilles, are all useful in suitable cases. The great benefit often to be derived from constitutional treatment, sea-bathing, and an outdoor life is not always sufficiently recognised.

Inhalations are of value when the irritation proceeds from the larynx, and the use of the bronchitis kettle and copious warm alkaline drinks are most valuable adjuvants in all conditions where the cough is aggravated by scantiness or viscosity of the expectoration.

"Ear" cough usually disappears immediately on the removal of the cause of the local irritation, often a plug of wax or a foreign body, such as a bead, inserted in the ear by the patient.

"Stomach" cough is to be treated by a suitable dietary, and the use of gastric sedatives such as bromine and hydrocyanic acid.

The barking cough of puberty is best treated by a plain but sufficient dietary, active outdoor exercise, early hours, and the use of tonics. Sir Andrew Clark was in the habit of ordering the syrup of the bromide of iron, with small doses of arsenic.

The danger attending the indiscriminate and injudicious use of sedatives and narcotics (especially opiates) in the treatment of cough, and the disastrous consequences which may result in young children and elderly patients especially, are pretty generally recognised. Nevertheless, it must not be forgotten that severe and prolonged coughing, coughing out of all proportion to the amount of the expectoration, if unrestrained, involves a great strain on the

lung, and may result in serious mischief to the lung tissue. Where a sedative is required small doses of chloral or of the bromides are often preferable to opiates. Children are not infrequently kept awake by an irritative cough which can be at once relieved by a few grains of chloral.

The bromides are of value in all forms of paroxysmal cough, and ammonium bromide has long been a favourite remedy in pertussis. Some cases of pertussis are greatly relieved by antipyrin in doses of about 1 gram for each year of the child's age.

In elderly patients a chronic cough associated with slight bronchial catarrh or congestion of the bases, and which has failed to benefit by the usual remedies, will often disappear under a course of digitalis in small doses. Strychnine is also of value in some of these cases.

**Coulomb.**—The quantity of electricity developed by a current of 1 ampere, the amount produced by an electro-motive force of 1 volt acting for 1 second against 1 ohm of resistance, the practical unit of electric quantity, named after Coulomb, a French electrician.

**Counter Irritation.**—The attempt to diminish some internal morbid action, such as inflammation of an organ, by applying irritation externally, "derivation by irritation." See CAUSTICS, CAUTERY.

**Counter Opening.**—A second opening made into an abscess cavity or other accumulation of fluid usually at a distance from the first opening, in order more easily and thoroughly to drain the cavity.

**Country Fever.**—A "continued thermic fever" occurring in the Carolinas, supposed to be due to the prolonged action of high temperatures, but often mistaken for malaria or typhoid fever, sunstroke (*q v*).

**Coup-de-soleil.** See SUNSTROKE.

**"Courses."** See MENSTRUATION AND ITS DISORDERS (*Terminology*).

**Court Evidence.** See MEDICINE, FORENSIC (*Post-mortem Reports and Examinations*).

**Courvoisier's Law.**—Concerns the state of the gall-bladder in common bile-duct obstruction. It is that, with some exceptions, when obstruction of the common bile-duct is due to calculus the gall-bladder is not enlarged, whereas in obstruction due to other causes, notably malignant disease, enlargement is the rule. Persistent jaundice, therefore, with a distended gall-bladder, according to this "law" indicates usually malignant disease.

**Couveuse.**—An apparatus, usually a

wooden or metal box, with appliances (hot bottles, or cavity containing hot water) for maintaining a constant temperature, used for the rearing of delicate or premature infants, an incubator. See PREMATURE INFANTS.

**Cowls.** See VENTILATION AND WARMING (Vacuum or Extraction Systems, Cords).

**Cowperitis.**—Inflammation of Cowper's glands (*q v*).

**Cowper's Glands.** See URETHRA, DISEASES OF (Anatomy, Male Urethra).

**Cowpox.** See VACCINATION.

**Cow-sheds.**—A cow-shed is any dairy in which milking cows are kept, it shall be sufficiently lighted and ventilated, and shall provide a minimum space of 800 cubic feet per cow, its drains shall be trapped, and it shall be provided with water-tap and hose for thorough and frequent cleansing, the floor shall be of an impervious material (concrete) and properly sloped and drained. The roof and walls shall be lime-washed (say, twice a year), and the shed should not be nearer a human habitation than 100 feet. See MILK (Industrial).

**Cow's Milk.** See MILK, INFANT FEEDING (Artificial), INVALID FEEDING (Milk and its Products), etc.

**Coxalgia.**—Pain in the hip, also hip-joint disease. See APPENDIX VERMIFORMIS, APPENDICULUS (Diagnosis), HYSTERIA (Hysterical Coxalgia), HYSTERIA (Infantile), LUMBAGO (Differential Diagnosis).

**Coxa Vara.**—A deformity due to malunion of the neck (or upper part of the shaft) of the femur, characterised by stiffness of the hip, limping, shortening of the limb, prominence of the trochanter and displacement of it above Nélaton's line, and diminution or loss of abduction. See DEFORMITIES (Leg and Thigh, Coxa Vara). *Coxa Valga* is also a deformity of the neck of the femur, but in this case there is loss of adduction. See also HIP-JOINT, INJURIES OF (Coxa Vara), HIP-JOINT, DISEASES OF (Diagnosis), RICKETS (Clinical Features).

**Coxitis.**—Inflammation of the hip-joint. See DEFORMITIES (Congenital Dislocation of Hip, Diagnosis), HIP-JOINT, DISEASES OF, RHEUMATISM, RHEUMATOID ARTHRITIS.

**Cracked—Pot Sound.** See BRUIT (Bruit de pot fêlé), PNEUMONIA, CLINICAL (Physical Signs, Red Hepatisation).

**Cramp.**—Spasm of a muscle or group of muscles, accompanied by pain, it is common as a nervous of profession or of employment, as writer's cramp (scrivener's palsy), pianist's cramp, tailor's cramp, shoemaker's cramp,

telegraphist's cramp, milkmaid's cramp, motorist's cramp, coachman's cramp, etc., etc. See ALCOHOLISM (Motor Variations, Spasm), CHOLERA, EPIDEMIC (Symptoms, Cramps), DIABETES MELLITUS (Nervous System), GOUT (Acute, Pseumotisms), GOUT (Triquetral, Nervous System), MUSCLES, DISEASES OF (Vascular Disturbance), SPASM (Physiology, General), STOMACH AND DUODENUM, DISEASES (General Symptomatology, Remote Symptoms), TETANY (Motor Symptoms), THYROID GLAND, MEDICAL (Exophthalmic Goitre, Nervous Symptoms).

**Crania Progenæa.** See CRANIUM PROGENIUM.

**Cranial Nerves.** See BRAIN, PHYSIOLOGY OF (Cranial Nerves), PHYSIOLOGY, NERVOUS SYSTEM (Medulla Oblongata and Cranial Nerves). See also NOSE (for First Nerve), RETINA AND OPTIC NERVE (for Second Nerve), OCULAR MUSCLES, AFFECTIONS OF (for Third, Fourth, and Sixth Nerves), FIFTH NERVE, AFFECTIONS OF, FACIAL NERVE, PARALYSIS (for Seventh Nerve), AUDITORY NERVE AND LABYRINTH (for Eighth Nerve), GLOSSO-PHARYNGEAL NERVE (for Ninth Nerve), VAGUS NERVE (for Tenth Nerve), SPINAL ACCESSORY NERVE (for Eleventh Nerve), HYPOGLOSSAL NERVE (for Twelfth Nerve).

**Cranio-**—In compound words *cranio-* (from *cranium*, skull) signifies relating to the skull (*Cranio-tomy*, for instance, is removal of a piece of the cranial vault to allow growth of the brain, *cranioclast* is a head-crusher and extractor, used in obstetrics, having a solid blade which is passed inside the cranium, and a fenestrated one which is applied outside (see LABOUR, OPERATIONS, *Embr-yotomy*), *craniometry* is the science of measuring crania, *craniopagus* is a teratological type of double monster in which the twins are united by the heads, the union being either by the occiputs (unopagus), or by the frontal regions (metopagus), or by the scapulae (acrocephalopagus), the *craniopharyngeal canal* is a passage leading (in the embryo) from the pharynx through the sphenoid bone into the cranium, and is a remnant of the diverticulum of the pituitary body, *craniostoma* is a defective (split) state of the cranium in which the brain is exposed, *craniostegosis* is contraction of the skull, *craniostosis* is premature ossification of the sutures of the cranial vault, *craniotabes* (or *craniomalacia*) is a morbid condition (rachitic) of the cranium in which the vault bones (especially the occiput) are thinned in places and give a sensation of yielding or crackling to the finger (see BONE, DISEASES, *Inherited Syphilis*, RICKETS, *Clinical Features*, *Head*), *craniotomy* is the operation of perforating the cranial vault so as to diminish the size of the cranium in difficult cases of labour (see LABOUR, OPERATIONS, *Embr-yotomy*).

**Cranium.** See BRAIN, SURGERY OF (*Trephining*), INSANITY, PATHOLOGY OF (*Pathological Anatomy, The Skull*), LABOUR, DIAGNOSIS AND MECHANISM (*Moulding of the Fetal Head*), PHYSIOGNOMY AND EXPRESSION (*Cranium*), SCALP.

**Cranium Progenium.**—Abnormal projection of the lower jaw, due to increase in its size or to defective growth of the upper jaw, with narrowing of the face, etc. See INSANITY, PATHOLOGY OF (*Skull*)

**Cransac.** See BALNEOLOGY (*France, Calcaevous*)

**Crapulence.**—Intemperance in eating or drinking, and the effects of such intemperance (Latin, *crapula*, intoxication, Greek, *κραπάλη*, a drunken headache)

**Crasis.**—Constitution or temperament (Gr *κράσις*, mixture)

**Crassamentum.**—The thick, jelly-like part of coagulated blood, consisting of the blood corpuscles and the fibrin, the clot (Latin, *crassare*, to thicken)

**Cratomania.**—A form of insanity characterised by the mania of power or superiority (Gr *κράτος*, strength, *μανία*, madness)

**Craw-craw.** See FILARIASIS (*Filaria Perstans*), SKIN DISEASES OF THE TROPICS (*Skin Diseases of Bacterial Origin, Crine-crave*)—A parasitic skin disease of West Africa (Dutch, *kraauwen*, to scratch)

**Cream.** See DIET (*Milk and its Products*), INFANT FEEDING (*Artificial Feeding, Cream*), INVALID FEEDING (*Food for the Aged, Milk and Cream*), MILK (*Dietetic*), PHYSIOLOGY, FOOD AND DIGESTION (*Butter and Cream*)

**Cream of Tartar.** See POTASH AND ITS SALTS (*Potassi Tartarus Acidus*)

**Creamery.** See MILK (*Industrial, Creamery*)

**Creat.**—Indian chmetta or *Andropogon paniculatus* See ANDROPOGON

**Creatin.**—An alkaloid or extractive (methyl-guanidin-acetic acid,  $C_4H_8N_4O_2$ ) occurring in muscle, and excreted in the urine in the form of *creatinin* ( $C_4H_7N_4O$ ), which is creatin which has lost a molecule of water. *Creatinæmia* is the morbid state ascribed to excess of creatin in the blood. See PHYSIOLOGY, TISSUES (*Chemistry of Muscle, Extractives*), PHYSIOLOGY, THE BLOOD (*Plasma and Serum*), PHYSIOLOGY, FOOD AND DIGESTION (*Flesh*), PHYSIOLOGY, EXCRETION (*Urine, Nitrogenous Substances, Creatinin*)

**Credé Method.**—(1) The Credé method of placental expression is carried out by seizing the fundus uteri (through the abdominal walls) with both hands and squeezing it firmly downwards and backwards towards the pelvic inlet (2) The Credé prophylactic treatment of the eyes of the child at birth consists in dropping one or two drops of a 2 per cent solution of silver nitrate into the conjunctival sac of each eye, in this way the risks of ophthalmia neonatorum have been greatly lessened, but it is not necessary always to use the silver nitrate solution, boracic lotion or distilled water may suffice

**Credulitas.**—The condition of being easily duped or persuaded, regarded as a variety of moria imbecilis or idiotism

**Creeping Eruption.**—*Larva migrans*, a skin eruption, characterised by a raised red line which may travel quickly over the body, due to the larva of the horse bot-fly (*Gastrophilus equi*), *dermatomyiasis linearis migrans æstiva*

**"Creeps."**—Extraordinary restlessness and irritability, with wakefulness, dry skin, thirst, and the sensation of fullness in the limbs, due to fatigue (bodily or mental), etc., the "hidgets" or dysphonia

**Cremaster.**—The suspensory muscle of the testicle, having (in the human subject) only slight power of drawing that organ up towards the inguinal canal, the name is derived from the Greek *κρεμᾶν*, to suspend

**Cremation.**—The reduction of the corpse to ashes by burning in a close furnace, this mode of disposing of the dead, which is preferable to earth-burial, if properly carried out, seems to have been first practised in Europe (in Italy) in 1869, the Cremation Society of England was formed in 1874 with Sir Henry Thompson as its first president, and, after some legal difficulties had been overcome, a crematorium was established at Woking and cremations successfully carried out, a Cremation Act was passed in 1902, and there are now crematoria at Glasgow, Manchester, Liverpool, Birmingham, Hull, Leicester, Hendon, and Darlington, as well as at Woking, the furnace used may be either reverberatory (a flame playing on the dead body) or regenerative (gas from coke being burned in the chamber), and the whole process can be performed in less than two hours, the cost is still considerable, but could be greatly reduced if the practice of cremation were common. It has been feared that this mode of getting rid of the dead body might check the detection of crime, but with proper precautions this is unlikely

**Cremometer.**—A graduated cylinder showing the percentage of cream rising spontaneously to the surface of the milk. See MILK (*Examination*)

**Cremora.**—Pharmaceutical preparations (for external use) containing glycerine or vaseline as a basis; creams (Latin, *cremor*, milk), such as *Hazeline Cream* (B W and Co)

**Creolin.**—A dark syrupy liquid, obtained from the dry distillation of coal, consisting largely of coal tar, fatty acids, and resins, and forming a white emulsion with water, it is germicidal and is therefore used as an antiseptic, it is contained in Jeyes' disinfectant

**Creosotal.**—Carbonate of creosote, an oil sometimes used in place of creosote and in the same dose, it is not so likely to produce indigestion. See CREOSOTE

**Creosote.** See BRONCHI, BRONCHIECTASIS (*Treatment, Creosote*), LUNG, TUBERCULOSIS (*Treatment, Creosote and its congeners*), PHARMACOLOGY, PRESCRIBING, TOXICOLOGY (*Organic Poisons, Creosote*)—Creosotum (Gr *σπῆς*, flesh, and *σείω*, to preserve) or creosote is an oily liquid, with a peculiar smell, obtained by the distillation of wood tar (*Pice Liquida*), and consists of a mixture of creosol ( $C_8H_{10}O_2$ ), cresol ( $C_7H_7O$ ), *oxyresol*, *methylcresol*, *guaiacol* ( $C_8H_7O_2$ ), etc. (Creosote from coal tar differs from the above in certain particulars) (Creosote is incompatible with oxide of silver, forming with it an explosive mixture. The dose of creosote is 1 to 5 m, and the drug is best given suspended in mucilage, as a pill, or in capsules (diluted with mineral oil). The official preparations are the *Mistura Creosoti* (dose,  $\frac{1}{2}$  to 1 fl oz) and the *Unguentum Creosoti*. The drug is useful in obstinate vomiting, in some forms of diarrhoea, in typhoid fever, and in fermentative dyspepsia, acting as an antiseptic, it is also slightly anæsthetic and styptic, and has been commonly used in toothache (cotton-wool plug soaked in it being applied to the tooth). Of late years it has been much lauded as a remedy in phthisis and bronchiectasis, as an inhalation

**Crepitus.**—The grating or crackling sound or feeling produced when the ends of a fractured bone are rubbed together (during manipulation). Crepitant râles are the crackling sounds heard over the lungs in pneumonia. See FRACTURES (*Methods of Examination*), PNEUMONIA, CLINICAL (*Physical Signs*)

**Crescent Bodies.** See MALARIA (*Malarial Parasite*)

**Cresol.**—Tolyl alcohol ( $C_7H_7OH$ ), one of the hydroxytoluenes, found in three isomeric forms (ortho-cresol, meta-cresol, and para-cresol). Cresolsulphonic acid ( $C_6H_4(SO_3H)CH_3$ ) is formed from it by the substitution of the molecule  $SO_3H$  for hydrogen.

**Crest.**—A ridge or crest or ridge-like structure, such as the crest of the ilium or of the tibia.

**Cresyl.**—The radicle of cresol ( $C_7H_7$ ), or methyl-phenyl. Cresol is regarded as cresylic acid, cresyl alcohol has the formula  $C_6H_4(OH)CH_3$

**Creta.** See CALCIUM AND ITS SALTS

**Cretan Fever.** See UNDULANT FEVER.

## Cretenism.

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See also HEAD (in *Cretinism*), INSANITY, ETIOLOGY OF (*Etiological Varieties*), THYROID GLAND, MEDICAL (*Thyroidectomy*), MENTAL DEFICIENCY (*Cretinoid Cases*), SLEEP, NORMAL AND MORBID (*Morbid Somnolence in Cretins*)

**DEFINITION.**—Cretinism (congenital myxædema) is the peculiar condition of arrested bodily and mental growth and development, with increasing deformity and debility, which results from a congenital deficiency of thyroid secretion due either to absence of the gland or to some morbid change interfering with its function

The name is also generally, and perhaps conveniently, applied to the cases where defective action of the thyroid begins in early childhood (juvenile myxædema)

**CAUSATION.**—That the essential cause of cretinism is deficiency of the thyroid secretion is now an accepted fact. The morbid influences, however, which lead to disease or atrophy of the thyroid gland, and thus abolish its secretion, are still to a large extent unknown. They are probably different in the endemic and sporadic forms of the disease

*Endemic cretinism* is generally met with in mountainous districts. It is always associated in distribution with endemic goitre, and a large proportion of deaf and dumb children are found in the same districts and families. The cause of these three conditions is evidently the same, and is known to be present in the drinking water. It is, in all probability, a micro-organism (Kochel), although this has not yet been absolutely proved

All inquiries into the primary cause of *sporadic cretinism* have hitherto had only negative results. The disease very often occurs in large and otherwise healthy families. Two or more cases are, however, sometimes found in one family, and it is also not very uncommon for cases of infantilism, achondroplasia, and other forms of dwarfing to be present among the brothers and sisters

In cases of *juvenile myxædema* there is often a history of one of the infectious diseases having occurred shortly before the symptoms were noticed

**DISTRIBUTION.**—Endemic cretinism is almost

unknown in Great Britain, but a few cases are to be met with, especially in Derbyshire, Somersetshire, Yorkshire, and Westmoreland. It occurs to a considerable extent in the mountainous regions of Europe, especially in Switzerland, France, and Italy, and has been reported as prevalent in various parts of North and South America, China, India, Madagascar, etc.

Sporadic cretinism is a comparatively rare disease, but cases of it have been reported from nearly every quarter of the world. There seems no reason to connect its occurrence with the physical characters of the district in which it is found.

**DESCRIPTION.**—*Infancy.*—Even in severe cases of cretinism there seems to be very little wrong with the child at birth. If the mother is observant, however, she soon notices that the baby is backward and apathetic—crying and laughing very little, and that the bowels are obstinately constipated, also often that the tongue seems too large.

On examining the infant cretin we are struck by his puffy, expressionless face and wrinkled forehead (Fig. 1), and on measurement he is found to be undergrown, although as yet there is no noticeable disproportion between the trunk, head, and limbs. His hands are characteristically broad and short. The temperature is subnormal. The thyroid is generally absent in sporadic cases, while in the endemic form there is usually a goitre.

*Childhood and Youth.*—As the child grows older his development lags behind, and the characteristic cretinous appearance becomes

the belly becomes more prominent, and marked lordosis develops. There is generally an umbilical hernia. Circumscribed fatty swellings appear above the clavicles and in front of the axillae. The dryness of the skin increases, and the hair is very scanty and dry. The fontanelle remains widely open. The milk teeth may or may not be delayed in appearance, but generally they remain too long in the gum, and they may all be present even as late as the eighteenth year. The child's growth and activity are greatly interfered with, so that at ten or twelve years old he often has the size and the feebleness of a boy of three or four. The mental condition in an ordinarily severe case of the disease is that of imbecility, but in slighter forms of the disease the child seems merely backward. He is dull and apathetic, slow of movement and of apprehension, but neat and tidy in his ways, and docile and quiet if not teased. Speech is generally long of being acquired, and the words used are few.

*Adult Age.*—In spite of their debility, cretins not infrequently live to forty, fifty, or even sixty years old. The adult cretin shows the disease in its most marked form (Fig. 2). He is generally about three feet in height, with large brachycephalic head, thick dry redundant skin, scanty coarse hair, and an open fontanelle. His trunk is stunted, and he has marked lordosis and a prominent belly; there is often also lateral curvature. The limbs are short and thick-set, with prominent hard muscles. The gait is very feeble and waddling. The extremities are always cold. There is little or no pubic or axillary hair. In the male the external genitals are like those of a child, while in the female the breasts remain quite undeveloped. Menstruation is very late of appearing and very irregular, if it comes at all. In the few cases in which pregnancy and delivery have taken place, the infant, although not myxœdematous, has been atrophied or hydrocephalic at birth, and has died soon after. The cretin's mental condition does not improve as age advances. Although very dull, he understands and observes more than he appears to, but he has little or no initiative. His interests are exceedingly limited. He is shy, morbid, and solitary.

During recent years a number of slighter cases have been described by Hertoghe and others as really instances of cretinism, although they lack many of the usual symptoms. In these there is little beyond stunted growth, delayed closure of the fontanelle, and some degree of mental dulness. The confirmation of the diagnosis is to be sought in the marked improvement which follows thyroid treatment.

**DIAGNOSIS.**—The two morbid conditions most apt to be mistaken for cretinism are achondroplasia and the so-called "Mongolian" type of imbecility.

The resemblance which achondroplastic dwarfs



steadily more striking. The myxœdematous swelling increases in the face and elsewhere,



have to cretins at birth is in some ways striking, but it is *adult* cretins they resemble. The extreme shortness of limbs which they exhibit is

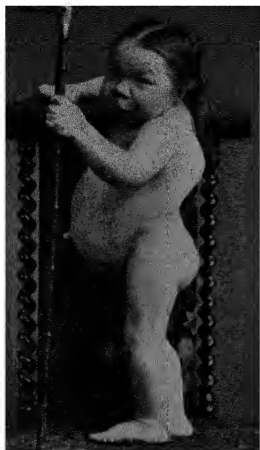


FIG. 2.

not a characteristic of cretin infants, while their soft natural skin and hair, their normal temperature and mental condition, and the peculiar formation of their hands (*see* "Achondroplasia") enable them to be readily distinguished.

"Mongolian" imbeciles resemble cretins in their backwardness, their frequently protruding tongue, scanty dry hair, and dwarfed stature. They differ in being less stunted in growth, in their physiognomy, in the shape of their hands, and in their general appearance and mental characteristics (*see* "Idiocy and Imbecility").

**MORBID ANATOMY.**—In the endemic form of the disease, goitre is present in about 75 per cent of the cases, while in the remainder the thyroid is absent.

In most cases of sporadic cretinism no trace of a thyroid can be discovered; in a few the gland is atrophied, and shows cirrhotic changes; rarely there is a cystic goitre.

**TREATMENT.**—The treatment of cretinism, like that of adult myxædema, consists in the continued administration of some preparation of thyroid gland by the mouth. The thyroid of the sheep is that generally used, and it may be given raw or in such form as the *Thyroidum Siccum* or *Liq. Thyroidæ* (B.P.) or various other dry preparations. One of the most reliable of these, and one which seldom disagrees, consists of the dried colloid matter of the thyroid (Hutchison). The dose must vary according to the age, size, and strength of the patient, and

according to the intervals at which the remedy is administered.

The fresh raw thyroid is probably more active and trustworthy than any of its preparations. It may be given to a young child, to begin with, in doses of  $\frac{1}{4}$  to  $\frac{1}{2}$  of a gland *twice a week*; or in double these doses to an older child or adult. After some time one whole lobe may often be taken each time with advantage. It is a curious fact that the improvement in cases where thyroid is given twice a week is just as continuous and satisfactory as that seen where the remedy is administered in small doses daily or every few hours.

Generally, however, it is more convenient to use one of the manufactured preparations, and to give it daily. In a young infant we may begin with gr. i. of the dried gland, or ii. of the *Liq. Thyroidæ*, or gr.  $\frac{1}{2}$  of the dried colloid matter. If these doses cause no unpleasant symptoms they may soon be increased. In adolescent or adult patients, from three to five times these doses may be given daily to begin with.

During treatment the patient may go about as usual, and no special diet is required. The temperature, weight, growth, and general health must be closely watched. Should the patient become feverish, and show signs of sickness, headache, etc., this indicates rest in bed and a diminution of the dose. Often after some weeks or months an increase of the dose is necessary to maintain the improvement satisfactorily.

In treating adolescent cretins it is very important to prevent, if possible, the bending of the legs which is apt to follow their greatly increased activity. For this purpose they should be made to lie as much as possible.

**THE RESULT OF TREATMENT.**—When the treatment is carefully carried out rapid and continuous improvement results. The temperature rises to and remains at the normal level. The unnatural swelling quickly disappears from the face and other parts of the body. The features lose their unnatural thickness and become more mobile, and the eyes look much brighter. At the same time the tongue ceases to be protruded, the voice becomes less guttural, and the child no longer snores at night. The abdomen diminishes greatly in circumference, and if an umbilical hernia is present it disappears. The fatty swellings also vanish at an early stage of the treatment.

The skin loses its harsh and dry feeling and becomes soft, and the cheeks show a natural flush. In young patients the hair falls out, at first, in considerable quantities, but is soon replaced by a new crop which grows more rapidly and is softer and often of a different shade of colour.

At first there is a considerable loss of weight, with a relaxed condition of the muscles and ligaments, but in the course of six months,

if not before, the patient gains flesh considerably. The limbs also become firm and strong, and the back straighter and more shapely. The retarded evolution of the teeth is actively resumed.

The growth of the skeleton is perhaps the most striking change of all. It begins at once and proceeds rapidly. The patient often gains 2 in height within the first two months, and may make as much as 6 or 8 in the first year. After that the rate of growth diminishes and approximates to the normal. The appetite is greatly increased by the treatment, and the bowels generally become regular in action. The mental improvement which occurs is apt, at first, to be greatly overestimated by the parents, because the child *looks* so much brighter and his movements are so much livelier than before. Within six months, however, there is unmistakable advance, and this continues and increases—the better-nourished brain becoming increasingly capable of work. The children become more inquisitive, more independent and enterprising, and more inclined to do things. They lose their shy, morose, self-centred disposition, and become bright, happy, childlike, and sociable.

In the milder cases, the arrears of bodily growth are soon fully made up, and the state of the intellect approaches, if it never quite reaches, the normal. In a certain proportion of cases, however, while the bodily recovery is more or less complete, the child remains an imbecile.

In adolescent cases (from sixteen to twenty-five years) the improvement is very great, but a considerable degree of deformity remains, owing to the lower limbs not growing in proportion to the rest of the body. There is also, at this age, a strong tendency for the legs to become much bowed, and this is extremely difficult to prevent.

In adults (after about thirty) the effect of treatment is much less. In them there is usually a growth of one or two inches during the first few months, and none after. The appearance of the face improves greatly, menstruation becomes more or less regular, and the breasts enlarge. Even at this age, however, the treatment is advantageous because of the great improvement which it causes in the patient's general health, and especially in his happiness. Although he still remains a child in mind and a dwarf in body, he begins for the first time to take an active pleasure in seeing and doing things and in associating with other people.

**Creyat.** See ANDROGRAPHIS

**Cribiform.**—Sieve-like (from Latin, *cribrare*, to sift), perforated with numerous small holes, e.g. the cribiform plate of the ethmoid bone.

**Cricoid.**—In compound words *cricoid* refers to the cricoid cartilage (Gr *κρίκος*, a ring, and *κίτος*, resemblance) of the larynx, e.g. *cricoid-arytenoid* (relating to the cricoid and arytenoid cartilages), *cricoid-hyoid* (relating to the cricoid cartilage and the hyoid bone), *cricotomy* (opening into the larynx by dividing the cricoid cartilage), etc. See LARYNX, AFFECTIONS OF THE CARTILAGES, PHONOLOGI, RESPIRATION (Voice).

**Criminal Responsibility.** See also UNCONSCIOUSNESS (*Double Consciousness*).—In holding a person responsible (i.e. liable to punishment) for his misdeeds the law rests on two assumptions or postulates regarding human beings. These are (1) that the individual can distinguish between "right" and "wrong" in the concrete case, and (2) that he is possessed of will-power adequate to control his impulses, and to control them in the light of that knowledge of right and wrong. The law *presumes* these capacities to be present—presumes a man to be *sane* according to this standard. Where, however, it can be established that, owing to mental infirmity, these capacities, or either of them, are lacking in any individual, the general rule of responsibility is relaxed. If a criminal act be unmistakably the result of *insanity* in the perpetrator, the accused is held to be not responsible therefor.

The decision of the question whether an accused person is, on the ground of insanity, to be exempted from responsibility for an act with which he is charged, is, of course, the function of the jury, under direction of the judge. But it is the duty of the medical expert to assist them in coming to a right verdict in the matter. It is the purpose of the present article merely to indicate, for the guidance of medical witnesses, the standpoint from which the matter will be investigated judicially by setting forth the generally accepted criteria of *legal* insanity. This article does not attempt to follow the controversy between medical and legal authorities as to what degree or what forms of mental derangement ought to exempt from responsibility for crime.

The authoritative statement of the law of England bearing on criminal responsibility is to be found in the answers of the supreme judges to the questions submitted to them by the House of Lords in 1843 (commonly known as "The Rules in MacNaughton's Case," 4 *St Tr* NS 847). The leading feature in these rules, as compared with the views previously enunciated by legal authorities in England, was the repudiation of a knowledge of right and wrong merely in the abstract as any test of sanity, and the substitution of the question whether the accused at the time of the act was labouring under such a defect of reason from disease of the mind as not to know the nature and quality of the *particular act* he was doing. Baron

Hume (in his Scottish treatise on *Crimes*, i. p. 37) had put the matter thus "The question must be relative to the particular act done, and the accused's knowledge of the situation in which he did it. Did he as at that moment understand the evil of what he did? Was he impressed with the consciousness of guilt and fear of punishment?" The English judges followed the Scottish authorities by laying it down that the inquiry must be directed to the accused's mental state in relation to the particular act done.

To establish a defence on the ground of insanity, therefore, it must be clearly proved that at the time of committing the act the accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing, or, if he did know it, that he did not know he was doing what was wrong. "If the accused was conscious that the act was one which he ought not to do, and if the act was at the same time contrary to the law of the land, he is punishable."

If a person under an insane delusion as to existing facts commits a criminal offence in consequence thereof, he is not necessarily freed from responsibility on the ground of insanity. Much depends on the nature of the delusion. The accused's responsibility is to be determined by considering whether, assuming as real the facts with regard to which the delusion exists, there would or would not be legal justification for his conduct. Thus, if under the influence of his delusion he supposes another man to be in the act of attempting to take away his life, and he kills that man, as he supposes, in self-defence, he would be exempt from punishment. If, on the other hand, his delusion was that a man had only injured him in reputation or fortune, and he killed him in revenge for such supposed injury, he would be liable to punishment. This statement of the law given by the English judges has not commended itself to medical men. As a test of responsibility it appears to be vitiated (as Dr. Maudsley points out) by the assumption that a man, having an insane delusion, has the power to think and act in regard to it *reasonably*, that at the time of the offence he ought to have and to exercise the knowledge and self-control which a sane man would, were the facts with respect to which the delusion exists real. It, however, stands as authoritative in the law of England, but, fortunately, it has not been very logically applied in actual cases. Its corrective is probably supplied by the more general rule of reference in each case to the accused's knowledge of right and wrong in regard to the particular act. In Scotland, on the other hand, it has been judicially recognised that a man may be entirely insane, and yet may know well enough that an act which he is doing is forbidden by

the law of the land. If a man have not a sane mind to apply his knowledge, the mere intellectual apprehension of an injunction or prohibition may stimulate his mind to do an act simply because it is forbidden, or not to do it because it is enjoined (Lord Moncrieff in case of Miller, 1874, 3 Couper's Reports, 16).

The "Rules in MacNaughton's Case" do not deal with the subject of *moral insanity*. For long the Courts rigidly refused to recognise such form of mental derangement. But *moral insanity* (meaning by that term mental derangement in which disorder of the moral faculties is more pronounced than disorder of the intellectual) is now undoubtedly accepted in the British Courts as freeing from responsibility or mitigating punishment according to the circumstances of the case. Further, *weakness of mind* of a lesser degree, not such as to free entirely from responsibility, is held either to warrant leniency in punishment, or, it may be, to justify a verdict of something less than the full crime charged, e.g. of manslaughter or culpable homicide instead of murder.

If insanity be proved, the question of responsibility is not affected by a consideration of what was the *cause* of the disorder. The fact that it was occasioned by accused's own acts (drunkenness or other excess) is not of consequence. Intoxication does not free from responsibility, insanity brought on by drunkenness will. When a person killed another under a "momentary hallucination induced by drunkenness," it was laid down in a Scottish case that the jury might acquit of murder and convict of the minor crime of culpable homicide (Robertson or Brown, 1886, 1 White's Reports, 93).

Presently existing insanity may bar criminal proceedings, before, at, or after trial. In such a case it is, of course, the *present* mental condition of the accused that the medical witness must speak to, and therefore an examination of accused ought to be made as shortly as practicable before the inquiry—on the same day, if possible. In Scotland, where insanity is pleaded in bar of trial, the facts to be established (before a judge without a jury) are that the accused is presently insane, and incapable of giving intelligent instructions for his defence.

**Criminology.**—The scientific study of the physical and psychical characters of criminals, criminal anthropology. See ABORTION (*Medical-Legal Aspects*), ANTHROPOLOGY, ANTHROPOMETRY, BERTILLOTTAGE, MEDICINE, FORENSIC (*Identity*), MEDICINE, FORENSIC (*Criminal Abortion*).

**Crisis.**—A paroxysm of pain (gastric, hepatic, nephritic, etc.) occurring during the progress of locomotor ataxia, or a sudden change in the course of life (e.g. at puberty or at the menopause), or during the advance of a disease

(e.g. rapid defervescence in the course of a fever). See LYSIS, PNEUMONIA, CLINICAL (*Clinical Features*), TABES DORSALIS (*Symptomatology Urethral, Bladder, Renal, Gastric, Intestinal, and Rectal Crises*), TEMPERATURE (*Fever, Defervescence*). Lumbar pain, in cases of movable kidney, has been termed Dietl's crisis (*qv*)

**Crispation.**—Feeble muscular twitches, the "fidgets" or "creeps"

**Crista.** See CREST

**Crocus.**—*Saffron* or *Crocus sativus*, and the stigmata and styles of *Crocus albus*, and contains crocin (a glucoside colouring matter) and a volatile oil, there is an official *Tinctura Croci* (dose, 5 to 15 m), and its chief use is as a colouring matter (e.g. in *Tinctura Cinchonæ Composita*). See PRESCRIBING

**Croft Spa.** See BALNEOLOGY (*Great Britain, Yorkshire, Sulphur Waters*)

**Cromble's Molar Ulcer.**—An ulcer which forms between the last two teeth in the upper or lower jaw in the early stages of sprue. See SPRUE (*Clinical Features*)

**Cross Birth.** See LABOUR, DIAGNOSIS AND MECHANISM (*Transverse Lies*), LABOUR, OPERATIONS (*Version*)

**Crossed.**—The name applied to some morbid state (e.g. paralysis) occurring in the opposite half of the body or of an organ to that in which the lesion producing it is. See BRAIN, TUMOURS (*Diagnosis, Regional, Pons*), HIP-JOINT DISEASES (*Bilateral, Crossed-Leg Deformity*), OCULAR MUSCLES, AFFECTIONS OF (*Paralysis, Double Vision*)

**Crotalidæ.**—The pit vipers. See SNAKE-BITES (*Classification*)

**Crotchet.**—A sharp hook on a stem, passed inside the perforated skull of the foetus (in embryotomia) and used as an extractor, "crotchet cases" are craniotomy cases. See LABOUR, OPERATIONS (*Embryotomy*)

**Croton Oil.** See also DERMATITIS TRAUMATICA ET VENENOSA (*Causal Agents, Vegetable*), DRUG ERUPTIONS (*Types, Papular, Vesicular, and Puscular*), PHARMACOLOGY, PRESCRIBING, TOXICOLOGY (*Abortifacients*).—A fixed oil extracted from the seeds of *Croton Tiglium*. It is brownish in colour, has a faint rancid odour, and is intensely irritating. Dose— $\frac{1}{2}$ –1 m. Preparation—Linimentum Crotonis. Externally croton oil has been used as a counter-irritant in diseases of the chest and in joint affections, but it causes severe pustulation and even skin destruction, and is now seldom employed. As a last resource it may be applied to destroy an obstinate patch of ringworm of

the scalp. Internally it has a violent purgative action and is usually given on a lump of sugar or mixed with butter. It is recommended in apoplexy and other conditions in which the patient is unconscious and unable to swallow, but it is preferable in the majority of such cases to administer jalap or other common purgative through a tube passed into the œsophagus.

**Croup.**—The name given, somewhat loosely, to laryngitis with or without fibrinous effusion, as well as to spasms of the glottis. See DIPHTHERIA, LARYNX, ACUTE AND CHRONIC INFLAMMATIONS (*Membranous Laryngitis*). The term *croupous* may be used in the above senses, it is sometimes employed also for any membranous exudation, diphtheritic or not. See CONJUNCTIVA, DISEASES OF (*Membranous Ophthalmia*)

**Crowborough.** See THERAPEUTICS, HEALTH RESORTS (*English, Sussex*)

**Crowning.**—The stage in labour when the vertex (or crown) of the head appears at the vulva, and is surrounded by the circle of soft parts (the labia and perineum) as by a crown. See LABOUR, DIAGNOSIS AND MECHANISM (*Vertex Cases, Extension of Head*). The covering of an old tooth with a gold or porcelain crown. See TEETH (*Bridge and Bar Work*)

**Crura.** See CRUS (plural, *crura*)

**Crus.**—The leg or thigh or any leg-like structure. Thus *cranial phlegmasia* is phlegmasia of the lower limb following thrombosis, the *crura cerebri* are the bundles of white substance which form the peduncles of the cerebrum, so also in *crura penis*, *crura clitoridis*, *crura cerebelli*, *crura diaphragmatis*, etc. The *cranial angina* of Walton is intermittent lameness due to arterio-sclerosis, it is the dyslasia angio-sclerotica of Eib.

**Crusta.**—A hard, dry formation forming an outer covering for the surface of the body, usually composed of the products of a skin disease, a scab, *crusta lactea* or "milk crust" is the scab often seen on the face of infants suffering from seborrhœa or eczema.

**Crutch.** See BEINHÄLFER

**Cruveilhier's Palsy.**—Progressive muscular atrophy or poliomyelitis anterior chronica. See PARALYSIS (*With Atrophy of Muscles*)

**Cry.**—As a sign of disease, more especially in young children, the cry has considerable diagnostic importance. See CHILDREN, CLINICAL EXAMINATION OF (*Respiratory System, The Cry*). The animal-like cry at the commencement of an epileptic fit (see EPILEPSY, "Epileptic Cry", HYSTERIA, *Diagnosis*) is very characteristic, the

"hydrocephalic cry" is heard typically in tuberculous meningitis and other cerebral diseases of children, the cries in a hysterical convulsion come on later than in an epileptic one (see HYSTERIA, *Hysterical Convulsions*)

**Cryaesthesia.**—A morbid degree of sensibility to cold (Gr *κρύος*, cold), the antonym is *cyanæsthesia*, a morbid degree of insensibility to cold

**Crymoses.**—Diseases ascribed to the action of cold (Gr *κρύος*, chill)

**Cryomotherapy.**—The use of cold as a remedial or therapeutic agent

**Cryoscopy.** See URINE, PATHOLOGICAL CHANGES IN (*General Properties, Freezing-point*) See also DECHLORINATION, IONIC ACTION, OSMOSIS, etc.—By cryoscopy (Gr *κρύος*, frost, and *σκοπεῖν*, to examine) is meant the determination of the freezing-point of a fluid. The method has chiefly been applied (in medicine) to the blood and urine, but any solution may be examined by this means, which affords a measure of the osmotic pressure. THEORETICAL.—According to Van't Hoff's *theory of solutions*, substances in solution behave like gases, the dissolved molecules exerting pressure on the walls of the containing vessel in their efforts to diffuse through as large a space as possible. This *osmotic pressure*, which can be measured directly by a manometer, depends (1) on the nature of the dissolved substance, (2) on the temperature, and (3) on the concentration. At the same temperature, the osmotic pressure of solutions of non-electrolytes is proportionate to the number of contained molecules, equi-molecular solutions exerting the same osmotic pressure, but in the case of electrolytes, such as salts, bases, and acids, which are partly dissociated into ions, the osmotic pressure is proportionate to the number of molecules + ions in solution. Thus a 5 per cent solution of sugar (non-electrolyte) exerts only half the osmotic pressure of a 1 per cent solution, but a 5 per cent solution of sodium chloride (electrolyte) exerts more than half the pressure of a 1 per cent solution, because in the latter case relatively more dissociation occurs in the weaker solution, and there are therefore relatively more ions than in the stronger. The similarity of the laws governing gases and solutions is shown in the following (Koranyi) —

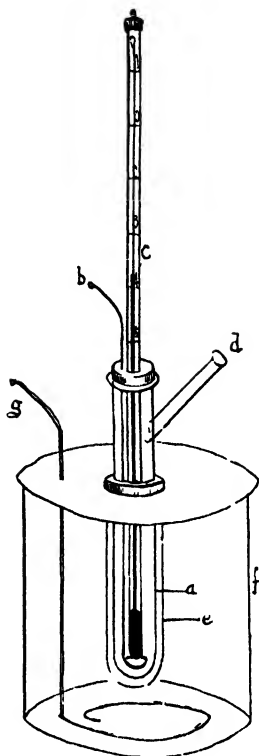
<i>Gases.</i>	<i>Solutions</i>
1. At a constant temperature the pressure is proportionate to the quantity of gas in a unit of volume	1. At a constant temperature the osmotic pressure is proportionate to the quantity of matter dissolved in a unit of volume, i.e. to the concentration

<i>Gases</i>	<i>Solutions.</i>
2. At a constant volume the pressure is proportionate to the absolute temperature, irrespective of the nature of the gas	2. At a constant concentration the osmotic pressure is proportionate to the absolute temperature, irrespective of the nature of the dissolved substance
3. At the same temperature and pressure, similar volumes of different gases contain the same number of molecules	3. At the same temperature and osmotic pressure solutions of different substances contain the same number of dissolved molecules in a unit of volume
When a gram-molecule of any gas occupies the space of 22.35 litres, it exerts at 0° C a pressure of 1 atmosphere	Any solution which contains one gram-molecule dissolved in 22.35 litres exerts at 0° C the pressure of one atmosphere
4. The pressure of a mixture of gases equals the sum of the pressures of all	4. The osmotic pressure of a solution of different substances is the sum of the osmotic pressures of all

The freezing-point of a solution is lowered, as compared with that of distilled water, proportionately to the number of molecules (or molecules + ions) it contains, hence from it the osmotic pressure can be estimated. Now a gram-molecule dissolved in 22.35 litres has an osmotic pressure of 1 atmosphere, hence a gram-molecule in 100 cc will have a pressure of 22.3 atmospheres, and it is found that this lowers the freezing-point by 18.5° C—in other words, a lowering of the freezing-point of the solution by 1° is equal to just about 12 atmospheres of osmotic pressure.

For practical purposes, since in medicine we are dealing with fluids containing dissociable molecules, and are concerned only with relative variations, we do not express the osmotic pressure in terms of dissolved molecules or in atmospheres, but take the freezing-point as a standard—the lower it is the greater the concentration of molecules or molecules + ions.

**Technique.**—Beckmann's cryoscope, or one of its modifications, is generally employed. It consists of a tube (a) which contains the fluid to be examined, and is closed by a doubly perforated cork through which a metal stirrer (b) and a thermometer (c) reading to  $\frac{1}{100}^{\circ}$  pass. The tube (a) has a lateral opening (d) and is enclosed in a second tube (e), the intervening air space acting as a non-conductor and preventing too rapid cooling. The whole is immersed in a larger vessel (f) containing freezing mixture, also provided with a stirring rod (g) and a cover (h). The thermometer employed has a range



of from  $-1^{\circ}\text{C}$  to  $1^{\circ}\text{C}$  or thereby, it must be carefully calibrated and requires to be standardized from time to time with pure distilled water which freezes at zero. The accuracy of its scale may be verified by a 1 per cent solution of pure sodium chloride, which freezes at  $-589^{\circ}\text{C}$ . Any errors detected must be allowed for in subsequently using the instrument. In making an observation the outer vessel is filled with a freezing mixture, which should not be too cold,  $-3^{\circ}$  being a desirable temperature. Five or ten cc of the fluid to be examined is now placed in the tube, which is then introduced into the freezing mixture. The fluid must be kept in gentle, constant motion during the observation. The mercury will be seen to sink steadily below the freezing-point, and then suddenly rises to a definite point at which it remains stationary. This is read off as the freezing-point. Crystals of ice begin to form, and the fluid eventually becomes solid, whereupon the thermometer falls again.

The whole manipulation is one of some

delicacy, and a number of precautions, as well as a certain amount of practice, are required to ensure reliable results. The bulb of the thermometer must be completely submerged without coming in contact with the tube. In stirring the fluid splashing must be avoided. For various reasons excessive cooling vitiates the results, hence the freezing mixture should not have a temperature lower than that mentioned. Excessive cooling may also be prevented by introducing a minute crystal of ice through the tube ( $d$ ) as soon as the temperature falls below zero, this accelerates freezing without diluting the fluid, as, of course, the ice introduced cannot melt at below  $0^{\circ}$ .

The freezing-point of human blood is extremely constant in health, lying between  $-55^{\circ}$  and  $-57^{\circ}\text{C}$ . It is customary to express this by the symbol  $\delta$ , the minus sign being omitted. The normal average freezing-point of the blood is written  $\delta 56$ . To denote the freezing-point of the urine  $\Delta$  is employed.

*Cryoscopy of the blood* is chiefly of importance as a gauge of the functional activity of the kidneys. It has the disadvantage that a fairly large quantity—10 to 20 cc—is required. In practice the osmotic pressure of the serum may be regarded as equal to that of the whole blood, hence the blood is allowed to coagulate and the serum alone is employed. A slight admixture of corpuscles is of no moment. So constant is the freezing-point in health that variations of  $0.1-0.2^{\circ}$  are pathological. Thus  $\delta = 58$  or  $59$  points to retention of molecules, while in some cases of anemia  $\delta = 65$  or  $7$ . In conditions of asphyxia the value of  $\delta$  is also raised, but falls to normal as soon as the blood is properly oxygenated. Putting this aside it may be said that a rise in the value  $\delta$  is a sign of renal inadequacy, and contra-indicates surgical interference in kidney lesions.

The information which cryoscopy of the blood affords in other directions is of minor import. In pregnancy  $\delta$  has a low value, rising to normal after delivery as the osmotic pressure of the blood returns. In hydremic conditions generally the freezing-point is high, in diabetes it is low, from concentration of the blood. It is possible that cryoscopy may yield forensic evidence of value, since in death from drowning  $\delta$  tends to approach the freezing-point of the fluid in which the body is immersed.

*Cryoscopy of the Urine*—The chief molecules influencing the freezing-point are those which are most abundantly present, viz. urea and sodium chloride. As dissociation of the latter takes place after the urine leaves the kidney the concentration of the urine cannot be taken as an exact measure of the osmotic energy of the organ. From the simple fact that  $\Delta$  varies widely in health, inferences must be drawn with caution, and as it is practically impossible to obtain the urine from each kidney separately for anything

but a short period, it will be seen that cryoscopy of the urine cannot be of such diagnostic importance as that of the blood. Sahli, indeed, believes that it presents few advantages over the estimation of the specific gravity, and states that in a urine free from sugar and protein  $\Delta$  may be calculated empirically by multiplying  $0.75^{\circ}\text{C}$  by the last two figures of the sp gr, carried to the third decimal place. In normal adults  $\Delta$  is greater than 8, averaging from  $1.2^{\circ}$  to  $2.3^{\circ}$  in the mixed twenty-four hours' urine. After copious draughts of water it may sink to  $1^{\circ}$ , or with restricted intake of fluid rise to  $3.5^{\circ}$ . In breast-fed infants  $\Delta$  is usually less than 8, varying from  $0.87^{\circ}$  to  $4.5^{\circ}$ . On the whole, in renal inadequacy  $\Delta$  is low, but of more importance is the fact a damaged kidney has lost its power of responding to the demands made on it, hence alterations of the intake of water are not followed by the normal corresponding variations in  $\Delta$ . The following figures from Koranyi illustrate this.—In a case of unilateral pyelonephrosis  $\Delta$  on the diseased side was  $4.9^{\circ}$ , on the healthy side  $1.63^{\circ}$ . After copious drinking, values of  $3.4^{\circ}$  and  $0.8^{\circ}$  respectively were given. Hence the freezing-point of the urine is a test of renal efficiency only when considered in relation to the fluid ingested, and a low value of  $\Delta$  is significant only when constant over considerable periods of time. If  $Q$  = the quantity of urine excreted in twenty-four hours,  $Q\Delta$  expresses the molecular excretion, and  $Q(\Delta - \delta)$  the osmotic energy in that time. A product equivalent to  $Q\Delta$  may be obtained by multiplying the last two figures of the sp gr by 2.33, and by the number of litres passed in twenty-four hours, which gives the approximate number of grams of solids excreted in that time (Sahli).

From the above it will be seen that while in cryoscopy of the blood we have a valuable method of estimating the activity of the kidneys, the same cannot be said of cryoscopy of the urine. In the present state of our knowledge the following general statements seem all that are warranted.— $\delta = 56$  shows that at least one kidney is adequate, and justifies surgical operation. If  $\delta = 59$  or more, nephrectomy is contraindicated, though less serious operations—*e.g.* nephrotomy—may be performed. If  $\delta = 6$  or more, it is probably unsafe to interfere until a more normal figure has been regained. When  $\Delta$  is persistently below 8 or 9 it indicates renal inadequacy, the urine of each kidney should be examined separately, or other tests (phloridzin or methylene blue) employed. A comparison of  $\delta$  and  $\Delta$  should be made. Some guidance as to the efficiency of the kidney function may be gathered from the variations of  $\Delta$  under copious drinking of water and restriction of fluid.

Cryoscopy of other fluids, pathological and physiological—*e.g.* the liquor amni and effusions—has been practised, but the results obtained,

though scientifically interesting, have had as yet no practical outcome.

**Crypt.**—A small cavity, or blindly-ending tube opening on a free surface (*e.g.* the crypts of Lieberkuhn in the intestinal mucous membrane). *Cryptitis* is inflammation of such a cavity or crypt.

**Crypto-**—In compound words *crypto-* (Gr. *κρυπτός*, hidden) signifies concealed or hidden.

**Cryptogenetic Septicæmia** (*Leuka*)—General septicæmia or blood-poisoning without any apparent local infection during life, and sometimes without any discoverable lesion after death, in many cases it is a terminal infection in individuals wasted by disease, but sometimes it affects persons apparently healthy, the streptococcus pyogenes is the commonest micro-organism found.

**Cryptomenorrhæa.** Absence of menstruation due to structural anomalies (*e.g.* imperforate hymen, vaginal atresia) which cause the retention of the menstrual blood, hidden menstruation. See MENSTRUATION AND ITS DISORDERS, Uterus, MALFORMATIONS OF (*Clinical Aspects, Symptomatology*).

**Cryptophthalmus.**—The teratological state in which the skin passes without a break from the forehead on to the cheek, entirely closing in the openings of the orbits, *ablepharon*, it is not to be confused with adhesion of the eyelids (*ankyloblepharon*), or with adhesion of the palpebral and ocular conjunctival surfaces (*symblepharon*).

**Cryptorchism.**—The teratological state in which one or both of the testicles is hidden, *e.g.* retained in the abdomen or arrested in the inguinal canal.

**Crystallina.** See SKIN, DISEASES OF SWEAT AND SEBACEOUS GLANDS (*Nidulamina*).

**Crystalline Lens.** See LENS, CRYSTALLINE, CATARACT, etc.

**Crystal Pox.** See VARICELLA.

**Crystals.** See CHARCOT-LÉYDEN'S CRYSTALS, FICES (*Abnormalities, Blood Crystals*), TEICHMANN'S CRYSTALS, etc.

**Cuban Itch.**—A mild form of smallpox, regarded by some as a new disease, Philippine itch.

**Cubebæ Fructus.** See also PHARMACOLOGY, PRESCRIBING, etc.—Cubebæ or the dried fruit of *Piper cubeba*, the fruits, which

have a warm, bitter taste, and an aromatic smell, contain a volatile oil (*Oleum Cubebe*, dose 5 to 20 m suspended in mucilage), an oleo-resin, cubebin ( $C_{10}H_{16}O_2$ ), cubelic acid ( $C_{12}H_{14}O_7$ ), and piperine, cubebs is given in doses of 30 to 60 gr., and the *Tinctura Cubebe* in doses of  $\frac{1}{2}$  to 1 fl dr, the chief action of the drug is a stimulant one upon the mucous membrane of the genito-urinary tract, but it is also a diuretic, and in small doses acts as a stomachic, it finds its greatest sphere of usefulness in gonorrhoea and cystitis, but it is also employed in bronchitis, pharyngitis, and asthma (as cigarettes). Lozenges of cubebs (*Trochisci Cubebe*) are official in the U S Pharmacopoeia.

**Cubital.**—*Cubital*, from *L. cubitus*, means relating to the forearm, or, more particularly, to the ulna, in the form of *cubito-* it enters into many compound words, such as cubito-carpal, cubito-radial, etc. See LAMPHATIC SYSTEM, PHYSIOLOGY AND PATHOLOGY (*Cubital Glands*).

**Cuca.** See COCA.

### **Cucurbitae Semina Præparata.**

—Melon pumpkin seeds (from *Cucurbita maxima*) or pepo, in doses of 3 to 4 oz, act as an anthelmintic in cases of tapeworm, they are given bruised with milk, and are followed by castor oil, they are official in the Indian and Colonial Addendum (1900) to the British Pharmacopoeia 1898.

**Culex.**—A gnat or mosquito. See FILARIASIS (*Filaria Bancrofti*), MALARIA (*History, Parasitology, The Mosquito*).

**Culicicide.**—Capable of killing gnats or mosquitoes.

**Cultivation.** See POST-MORTEM METHODS (*Bacteriological Investigations, Cultivation*), SKIN, PARASITES (*Method of Cultivation*).

**Culture.**—A growth (eg of micro-organisms) due to cultivation, a pure culture being a growth in which there is only one micro-organism.

**Cumin Fruit.**—The seeds of *Cuminum cyminum*, aromatic in character, from them is got *cumic aldehyde* or *cuminol* ( $C_{10}H_{12}O$ ), and from it *cumic acid* ( $C_{10}H_{12}O_2$ ) and *cumin alcohol* ( $C_{10}H_{14}O$ ).

**Cumulative Action.** See PHARMACOLOGY, TOXICOLOGY. —When symptoms of poisoning are suddenly developed in an individual who has been for some time taking a drug without any evil effects, these symptoms are ascribed to what is called cumulative action, thus, strychnine may be taken for a time in small doses without producing any unpleasant effects, and then, without warning, it may cause

spasms, this action may be caused by rapid absorption due, for instance, to some transitory state of the bowel, or by diminished excretion of the drug (eg by the kidneys).

**Cuneate Lobe.** See BRAIN, PHYSIOLOGY (*Median Aspect of Cerebral Hemisphere, Cuneus*), PHYSIOLOGY, NERVOUS SYSTEM (*Cerebrum, Localisation of Functions, Visual Centre*).

**Cuneohysterectomy.**—The removal of an elliptical (wedge-shaped, from *cuneus*, a wedge) piece of tissue out of the uterine wall (convex side) at the site of the angle of flexion, and the suturing together of the margins of the wound, the operation is performed by abdominal section in cases of persistent and otherwise incurable cases of uterine flexion (retro- or ante-flexion) in order to correct the flexion.

**Cupping.**—An operation in which cupping-glasses are applied to the skin in order to determine an excessive flow of blood to any part thereof (*dry-cupping*), this is done by raising the air in the cup (by heat or by an air-pump), and the effect may be increased by scarifying the skin (*wet-cupping*) and so withdrawing some of the blood.

**Cupping of the Optic Disc.**—There is a depression visible about the centre of the optic disc of the eye, and this is known as the "physiological cup", but under continuous pressure (eg in glaucoma) a "pressure excavation" or pathological degree of cupping takes place, recognisable on ophthalmoscopic examination. See GLAUCOMA (*Symptoms, Excavation of the Optic Disc*), RETINA AND OPTIC NERVE (*Anatomy*).

**Cuprum.** See COPPER.

**Curara.**—Curara, known also as OMARI, WOURARI, WOUARI, UARI, and CURAIE, is a South American arrow-poison, it is prepared from decoctions and extracts of various plants (an *Atum*, various species of *Stychnos*, etc), its powerfully poisonous effects are due to the alkaloid *curarina* ( $C_{12}H_{15}N$ ) which it contains, it paralyses the peripheral ends of the motor nerves of the voluntary muscles, and has therefore been used in cases of tetanus, it is not official, but there is an *Injunctio Curaræ Hypodermica* (dose, 1 to 6 m subcutaneously). See ALKALOIDS (*Curarina*), ELECTRICITY (*Paralysis, Curarised Muscles*).

**Curd Soap.**—Sapo Animalis or Curd Soap is chiefly stearate of sodium, and is used as a basis, being contained in *Extractum Colocyntidis Compositum*, *Pilula Scammonii Composita*, and *Limentum Potassii Iodidi cum Sapone*. See PRESCRIBING.

**Cure.**—The word *cure*, although generally signifying a return to health or the means by which it is accomplished, has also the special



meaning of a particular course of medical or surgical or hydropathic treatment, thus, there is the Banting Cure for Obesity (*q v*), the Gold Cure for Alcoholism (a secret method), the Grape Cure for Habitual Constipation, the Kneipp Water Cure (walking barefoot through meadows in the dewy morning), the Schott Cure (muscular movements and Nauheim baths) for Heart Disease, etc

### Curettage, Uterine.

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See also ABORTION (*Treatment, Incomplete Abortion*), ATROPHICUS, GYNECOLOGY, DIAGNOSIS IN, PELVIS, PERINEUM AND PELVIC FLOOR (*Prolapsus Uteri, Treatment*), UTERUS, INFLAMMATIONS OF (*Chronic Endometritis*), VAPORISATION, ZESTIOKAUSIS

It is fifty-six years since Recamier advised the scraping of the inside of the uterus with a sort of scoop with subacute edges in cases of metrorrhagia from "intra-uterine fungosities", but discouraging and even fatal results followed this first use of the curette, and it was not till 1865 that Marion Sims ventured to characterise the operation as a practice "now recognised as legitimate". Sims' curette was a sharp one, and so was the spoon curette of Simon introduced in 1872, but the blunt instrument invented by Thomas, and popularised in this country by Mundé, by its almost complete safety did a great deal to establish the curette in the favour of the profession. Its chief use was as a means of diagnosis. Since 1878 the sphere of usefulness of the uterine curette has been enormously widened, sharp and blunt and flushing instruments are employed, and the operation of curettage is looked upon as one requiring care and skill, and not simply as a trifling diagnostic method which could be safely carried out on the consulting-room couch.

The INDICATIONS for curettage may be divided into therapeutic and diagnostic, and of these the former are nowadays regarded as the more important, and will be considered first.

*Therapeutic Uses*—(1) *Hæmorrhage* from the genital organs is one of the commonest and most fully established uses of the curette. The bleeding may be from the cervix uteri, or even from the vaginal walls, as in cases of malignant disease of these parts, and then the curette acts only as a palliative, in the great majority of cases, however, the hæmorrhage is from the body of the uterus, and curettage is at any rate expected to be curative. Uterine corporeal hæmorrhage may be due to retained products of conception, as after an incomplete abortion, then the cavity may be cleared out with the index finger, which is, under the circumstances, the best curette, but if the cervical canal has

closed and the bleeding continues, notwithstanding the lapse of some days or weeks, then dilatation and a thorough curetting of the interior will be needed, for decidua remains are often very firmly fixed, and need not be of much size in order to set up a great amount of hæmorrhage. Again, the hæmorrhage may be due to chronic non-septic hyperplastic endometritis, and in such cases the removal of the greatly thickened endometrium by the curette (blunt or sharp) with previous dilatation of the cervix will often give immediately satisfactory results, especially if the curettage be followed by a thorough cauterisation of the uterine interior. It cannot be said that hæmorrhage from a uterus affected with fibroid tumours has been more than temporarily checked by curettage, for the mucous membrane under these circumstances is not always hypertrophied (it may indeed be atrophic), and the congestion may be seated in the muscular coat, which is of course beyond the reach of the scraping. In the case of sarcomata and carcinomata of the body of the uterus the curette is only a palliative means of treatment, but it has a value as a diagnostic, and, further, it may be used as a preliminary procedure before the performance of vaginal hysterectomy for these uterine tumours.

(2) *Septic and other infected states* of the uterus, and even of the Fallopian tubes, have during recent years been treated by curettage, but all gynecologists are not agreed as to the value of this indication, and, of course, all infected states are not equally amenable to curettage. In acute septic endometritis, for instance, following abortion, or even labour at the full term, the curette must be used only after consideration, and not as a routine plan of treatment, but even under these conditions it has its uses, especially if intra-uterine douching, iodoform packing, and the antistreptococcal serum fail to reduce the temperature. Great care must be taken not to perforate the soft walls of the puerperal or post-abortum uterus. In chronic infected states in which the uterus is enlarged and displaced, menstruation disordered, and purulent and profuse leucorrhœa in existence, the curette often gives the best results. Even in cases in which the appendages are infected it has been recommended that the uterus be curetted in order to drain pus-containing tubes through it.

(3) *Dysmenorrhœa and sterility* form yet other indications for curettage, but it is often difficult to separate those from the chronic infected states. At any rate it is probable that the straightening of the uterus and the dilatation of the cervical canal may be as efficacious as the curettage in the improvement which undoubtedly sometimes follows. (4) In the *induction of abortion* for dangerous conditions in pregnancy, such as incoercible vomiting, the curette (preferably the blunt variety) may be employed, especially when

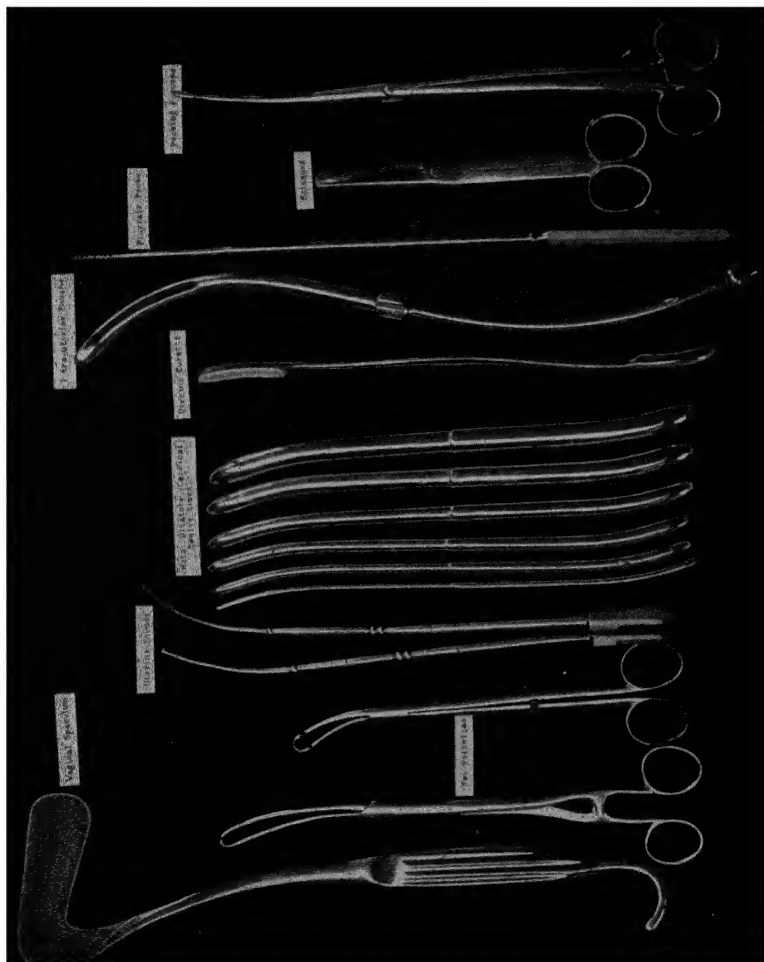
it is important to carry out the induction rapidly and with small loss of blood (5) Curettage may be used as a *prophylactic and preliminary procedure* when it is intended to operate on the vaginal walls or cervix, or to do hysterectomy, in order to prevent the infection of the wounded surfaces with septic material, etc., from the uterus.

The *diagnostic uses* of the curette consist in the removal by this means of scrapings of uterine mucous membrane and then examination under the microscope in order to separate conditions of simple endometritis from sarcoma and carcinoma, and even from fibro-myoma of the uterus.

**THE TECHNIQUE**—When the condition calling for curettage permits the fixing of dates beforehand, the time chosen should be the week after the menstrual period. On the day before operation the patient should keep in bed, her urine should be tested, and some opening medicine should be given, to be followed by an enema next morning. The vulva and vagina should be thoroughly scrubbed out, and the operator and assistant should take the same personal precautions to ensure surgical cleanliness as if a laparotomy were anticipated. The armamentarium consists of a curette, which may be either Martin's sharp curette (which closely resembles that recommended by Récamier), or an instrument with a hollow stem, by means of which a stream of antiseptic solution can be sent through the uterus (flushing curette), a vaginal speculum, such as Simon's, two or more strong-toothed volsellæ with clips, a set of cervical dilators, such as Hegar's, three or four uterine sounds or Playfair probes armed with cotton-wool, a male catheter, No 10, an intra-uterine douche and a pair of curved packing forceps, some pledgets of gauze, and iodoform gauze in strips for packing. There should be at hand a bottle of iodised phenol for applying to the interior of the uterus on the armed sounds, if regarded necessary, there must, of course, be an anæsthetic (chloroform), and an antiseptic solution (perchloride of mercury, 1 in 5000). The patient, having been anæsthetised, should be placed in the lithotomy position on a table in a good light. The field of operation should be protected with sterilised towels, and the hips should be raised upon a pad of antiseptic cotton covered by Mackintosh. The external genitals and vulva should be again thoroughly washed and scrubbed, and the urine drawn off. The speculum is then passed and held in position exposing cervix and vaginal vault, and the operator seizes and draws down the cervix by means of the volsellæ, one being attached to the anterior and the other to the posterior lip. If the uterus be fixed by pelvic adhesions this should not be attempted. The dilatation of the cervical canal is now begun, unless it is already sufficiently open to allow the passing of the curette, the Hegar dilators, which have been carefully sterilised, are now slowly

introduced one after another, beginning with perhaps a No 3 and working up to a No. 13 or 14, and always allowing a minute or two to elapse between successive ones. From fifteen to twenty or even thirty minutes should be allowed for this part of the operation. Each dilator should be warmed and oiled before introduction. The curette is now passed into the uterus and the walls systematically scraped with it (first anterior, then posterior, and so on), the scraping, which is accompanied by a creaking ("le cri uterin"), being controlled by the left hand placed over the abdomen. The contents of the curette should be floated off into a cup of clean water for future microscopic examination. Next, the uterine cavity may be washed out with an antiseptic solution, and iodised phenol applied to the interior on sounds or Playfair probes if the caustic effect be desired. Finally, the uterus and vagina should be packed with a long strip of iodoform gauze, a pad of antiseptic absorbent wool should be placed over the vulva, and the whole held in position with a bandage. A hypodermic injection of ergotin (3 gr.) may be given to aid uterine contractions. The patient is then put back to bed and kept quiet. The urine may require to be drawn off, but it is often passed naturally. If the curettage has been done for suppurative conditions, the packing may need to be frequently changed, if not, it may be allowed to remain in for two or three days. The patient should be kept in bed for five days or a week, for longer if the operation has been performed for grave conditions, she ought to rest in bed at the next menstrual period, as the discharge is sometimes excessive then, and marital relations should not be resumed for eight weeks in order to permit of the complete restoration of the endometrium. It has to be borne in mind that curettage does not prevent future conception, it has indeed cured sterility.

The **DANGERS** of uterine curettage, if carried out with surgical cleanliness, are not many or great. Occasionally perforation of the uterine wall has occurred, especially when the operation has been done in the puerperium, then if grave symptoms, such as hæmorrhage or prolapse of intestine, appear, it may be necessary to perform hysterectomy, but in many cases no harm has resulted. Sepsis is not a common danger nowadays when curettage is looked upon as an operation and not as a diagnostic method to be done in the consulting-room or with the patient in bed. Abortion may result through want of diagnosis of pregnancy, therefore it is always well to inquire into the menstrual history, and to make a careful bimanual examination before beginning the cervical dilatation. Complete obliteration of the uterine cavity has in a few instances followed curettage, possibly on account of a sort of superinvolution. Rupture of purulent collections in the pelvis is a danger which



CURETTAGE



used to be much feared, and tubal disease and perimetritis were formerly regarded as CONTRA-INDICATIONS to curettage, but the risk has probably been exaggerated, and it is now held by some gynecologists to be good treatment to use the curette in those very conditions which were regarded as contra-indications. Of course under these circumstances the alternatives are vaginal hysterectomy or laparotomy. Pregnancy is a certain contra-indication, save when it is desired for some good reason to induce abortion.

**Currents.** See PHYSIOLOGY, THE TISSUES (*Muscle, Electrical Changes*)

**Curschmann's Spirals.** See ASTHMA (*Symptoms, Sputum*), BRONCHITIS (*Symptoms, Casts*), EXPECTORATION (*Microscopical Examination of Sputum, Curschmann's Spirals*)

**Curtilage.**—In sanitary law *curtilage* is defined as "a courtyard, backside, or piece of ground lying near to a dwelling-house"

**Curvature.** See SPINE, SURGICAL AFFECTIONS OF (*Lateral Curvature or Scoliosis*), STOMACH, DISEASES OF (*Anatomy, Curvature*)

**Curve.** See CARL'S, CURVE OF, EMBRYOLOGY (*Curves*), GENERATION, FEMALE ORGANS OF (*Pelvis, Curves*), LABOUR, OPERATIONS (*Forceps*). PHYSIOLOGY, CIRCULATION (*Cardiac Cycle, Graphs Curves*)

**Cusco's Speculum.** See GYNECOLOGY, DIAGNOSIS IN (*Vaginal Specula*)

**Cusp.**—A pointed end, projection, or extremity, e.g. of the crown of a tooth (TEETH, *Anatomy*), or of the valves of the heart (PHYSIOLOGY, CIRCULATION, *Heart, Valves*)

**Cuspariæ Cortex.** See also CARMINATIVES, PHARMACOLOGY, etc.—*Cusparia Bark* or *Angustura Bark* is the dried bark of *Cusparia febrifuga*, it has a disagreeable odour and a bitter aromatic taste, it contains several alkaloids (e.g. *cusparine* or *angustinine*,  $C_{20}H_{19}NO_9$ , *galpine*,  $C_{20}H_{21}NO_9$ , and *cusparidine*,  $C_{19}H_{17}NO_9$ ), a bitter principle (*angosturine*), and an aromatic oil, its official preparations are *Tinctum Cuspariæ* (dose, 1 to 2 fl oz), and *Liquor Cuspariæ Concentratius* (dose,  $\frac{1}{2}$  to 1 fl dr), its action is that of a stomachic and carminative (like *Calumba Root*), it is used to make *Angustura Bitters*, and it has been used as a febrifuge (in S America, whence it is obtained)

**Cusso.**—*Cusso* or *koussou* consists of the dried panicles of an Abyssinian plant of the order of the Rosaceæ (*Brayera anthelmintica*), and it is occasionally used as an anthelmintic, it contains an active resinoid principle, koussin ( $C_{31}H_{48}O_{10}$ ), soluble in alkalies, along with tannic

acid, an oil, etc. See ANTHELMINTICS, PARASITES (*Cestodes, Trina Solium*)

**Custard.** See INVALID FEEDING (*Prepared Foods, Diet during Convalescence*)

**Cut Throat.** See NECK, REGION OF (*Cut Throat*), MEDICINE, FORENSIC (*Suicide*)

**Cutaneous Diseases.** See DERMATITIS, SKIN, MYIASIS, etc

**Cuté.** See CARATÉS, PINTA

**Cuticle.**—The epidermis or scarf skin. See PHYSIOLOGY, TISSUES (*Epithelium, Stratified Squamous*), SKIN, ANATOMY AND PHYSIOLOGY

**Cutis.**—The skin, especially the corium or derma, *cutis anserina* or goose-skin is that state of the integument when from emotion or cold the hair follicles are everted and form projections on the surface, *cutis lassa* is a loose condition of the skin, dermatolysis, and *cutis testacea* is a name for *ichthyosis neonatorum*

**Cuvier, Duct of.**—In the embryo the cardinal veins (anterior or jugular and posterior) unite to form the duct of Cuvier which carries the blood to the sinus venosus which opens into the auricle of the heart. See EMBRYOLOGY, HEART, EMBRYOLOGY

**Cyan- or Cyano-**—In compound words (*cyan-* or *cyano-* from Gr *κυανος*, dark blue) means either blue in colour or else relating to the chemical compound cyanogen ( $C_2N_2$ ), for instance, *cyanosis* (*vide infra*) means blueness of the skin, *cyanuric* is a blue deposit found occasionally in the urine, while a *cyanide* is a compound of cyanogen with a metal or an organic radicle

**Cyanate.**—A salt of cyanic acid ( $HCNO$ ), such as potassium cyanate ( $KCNO$ ), or lead cyanate ( $Pb(CNO)_2$ ), or ammonium cyanate ( $NH_4^+NO^-$ ), which Wohler (in 1828) succeeded in transforming by evaporation into urea ( $CON_2H_4$ ), an isomeric form

**Cyanic Acid.**—A colourless volatile liquid ( $HCNO$ ), producing a caustic effect, and forming salts (cyanates) with metals

**Cyanide.**—A compound of cyanogen ( $C_2N_2$  or  $Cy$ ) with a metal or an organic radicle, e.g. potassium cyanide ( $KCN$ ), potassium ferrocyanide ( $K_4FeC_6N_6$ ), mercuric ( $Hg(CN)_2$ ) or silver cyanide ( $AgCN$ ). See DERMATITIS TRAUMATICA ET VENERATA (*Special Eruptions*), TOXICOLOGY.

**Cyanogen.**—A compound radicle ( $C_2N_2$ ), which can be isolated, by heating mercuric cyanide, as a colourless gas burning with a peach-blossom coloured flame. See TOXICOLOGY (*Cyanogen Compounds*)

**Cyanosis.**—The morbid condition in which the skin has a bluish colour, due to imperfect aeration of the blood on account of congenital malformations of the heart (*morbus caeruleus*) or of asphyxia or collapse. See BRONCHI, BRONCHIAL GLANDS (*Enlarged Glands, Symptoms and Signs*); BRONCHI, BRONCHITIS (*Insanity of Cyanosis*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Effects of Cardiac Disease*); HEART, CONGENITAL MALFORMATIONS OF (*Physical Signs*); RAYNAUD'S DISEASE; TOXICOLOGY (*Nitro-benzene, Aniline*).

**Cycle.**—The period of time during which certain events occur in a definite sequence; usually one cycle is followed by another in which the same events occur again in the same order. See HEART, PHYSIOLOGY OF (*Cardiac Cycle*); MENSTRUATION; PHYSIOLOGY, CIRCULATION (*Heart, Cardiac Cycle*).

**Cyclic Albuminuria.**—A morbid state of the urine, in which albumen is found, but only at certain hours of the day. See URINE, PATHOLOGICAL CHANGES IN (*Albuminuria, Significance of*).

**Cycling.** See ANKLE-JOINT, REGION OF, INJURIES (*Sprain, After-Treatment*); APPETITE (*Loss of, Treatment*).

**Cyclitis.**—Inflammation of the ciliary body of the eye. See GLAUCOMA (*Causes of Secondary*); IRIS AND CILIARY BODIES (*Inflammatory Conditions, Cyclitis*); SYPHILIS (*Tertiary, Eye and its Appendages, Cyclitis*); TYPHOID FEVER (*Ocular Complications*).

**Cyclocephalus.**—The teratological state of the face in which there is a single median eye, or in which two eyes (separate or fused) lie in one median orbital cavity; a nasal proboscis above (or below) the median eye may or may not be present.

**Cyclone.** See METEOROLOGY (*Winds, Cyclones and Anti-Cyclones*).

**Cycloplegia.**—Paralysis of the ciliary muscle. See EYEBALL, INJURIES OF (*Contusion, Cycloplegia*); IRIS AND CILIARY BODIES (*Injuries, Traumatic Mydriasis*).

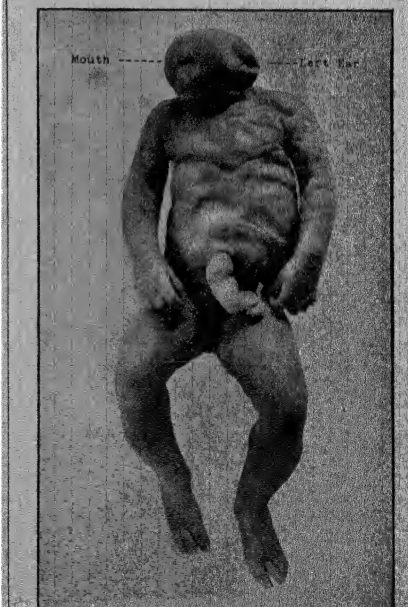
**Cyclopia.**—The single-eyed monstrosity (*synopsia*), in which the two orbits and their contents are more or less completely fused together in the middle line of the face, so named after the one-eyed Cyclops (*Polyphemus*) of mythology; there is usually a nasal proboscis or tube above the single orbit, and there are commonly four eyelids; the brain is markedly malformed, the corpus callosum, falx, septum lucidum, olfactory lobes, and optic nerves being

often absent. See CEBOCEPHALUS; CYCLOCEPHALUS;



CYCLOPTIA; TERATOLOGY (*Malformations of the Cranium and Face*).

**Cyclotia.**—The teratological type in which



median double eye with absence or defective development of the lower jaw and approximation of the external ears below the defective face; cyclops hypo-agnathus (*Taruffi*). In the accompanying illustration the single eye (on the top of the head) is not shown. See CYCLOPIA; etc.

**Cyesis.**—Pregnancy (Gr. *κύσις*, pregnancy or conception); *pseudocyesis*, therefore, is spurious pregnancy, and *cyesognosis* is the diagnosis of pregnancy.

**Cylindroma.**—A variety of epithelioma, containing cylindrical hyaline bodies, occurring most often on the face.

**Cyllosomus.**—A teratological type in which there is lateral exentration in the lower



part of the abdomen with absence or incomplete development of the lower limb of the same side (Gr. *κυλλός*, crippled, and *σώμα*, the body).

**Cymene.**—One of the benzene series of hydrocarbons; it is really methyl-isopropylbenzene ( $C_{10}H_{14}$ ); and it occurs in several volatile oils (e.g. oleum eucalypti and oleum eucali).

**Cynanche.**—Acute inflammation of the throat or of the neighbouring parts, causing difficulty of breathing and of swallowing, sometimes with protrusion of the tongue (Gr. *κύων*, a dog, and *ἄγχειν*, to strangle); there are different varieties of it, such as cynanche parotidea (mumps), cynanche laryngea (croup), cynanche

thyroidea (goitre), cynanche tonsillaritis (quinsy). See ANGINA, and under the various diseases (LARYNX, ACUTE INFLAMMATION; etc.).

**Cynanthrophia.**—That form of insanity in which the subject behaves like a dog (barks, runs, etc.), imagining himself to have been changed into one.

**Cynic Spasm.**—The contraction of the facial muscles by which the teeth are shown (as in a snarling dog), by the approximation of the angle of the mouth to the outer canthus of the eye.

**Cynobex Hebetis.**—The barking cough of puberty, a peculiar convulsive or spasmodic cough occurring mostly in boys about the age of puberty. See HYSTERIA; SPASM (*Varieties*).

**Cynocephalus.**—The teratological type in which the head of the foetus resembles that of a dog; it is generally the result of anencephalus or exencephalus.

**Cynolyssa.**—Rabies (*q.v.*).

**Cynorexia.** See BULIMIA.—A voracious appetite; “hungry as a dog.”

**Cyotocia.**—Parturition (Gr. *κύος*, ovium, and *τόκος*, birth).

**Cyphosis.** See KYPHOSIS; SPINE, SURGICAL AFFECTIONS (*Angular Curvature*); etc.

**Cyprus Fever.** See UNDULANT FEVER; MALTA FEVER.

**Cyrtometer.**—An instrument for measuring the curve of any part of the body (Gr. *κυρτός*, curved, and *μέτρον*, a measure), especially of any part, such as the chest, in which the curve is constantly varying; the curved tracing obtained is a *cyrtograph*. See PHYSIOLOGY, RESPIRATION (*Movements of Chest*).

**Cyst.**—A morbid structure, consisting of a wall or sac and contents of varying nature in a liquid or semi-solid state (Gr. *κύστις*, bladder). See BONE, DISEASES OF (*Cysts*); BRAIN, CYSTS AND CYSTIC DEGENERATION; BRAIN, SURGERY OF (*Trephining, Cyst of the Brain*); JOINTS, DISEASES OF (*Tumours and Cysts*); KIDNEY, SURGICAL AFFECTIONS OF (*Cysts*); KIDNEY, SURGICAL AFFECTIONS OF (*Hydatid Cysts*); LARYNX, BENIGN GROWTHS OF (*Cystoma*); LIVER, DISEASES OF (*Cysts*); MAMMARY GLAND, DISEASES OF (*Cysts, Galactocele, Multiple, Serous, Hydatid*); MAMMARY GLAND, DISEASES OF (*Neoplasms, Cysto-Adenoma*); MEDIASTINUM (*Certain Tumours, Dermoid, Congenital, and Hydatid Cysts*); NECK, REGION OF (*Cysts and Cystic Tumours*); OVARIES, DISEASES OF (*Cystic Tumours, Broad Ligament*).

*Cysts, etc.)*, PALATE (*Tumours, Dermoids, Cysts*), PANCREAS, DISEASES OF (*Cysts*), PERITONEUM (*New Growths, Cysts*), PERITONEUM, TUMOURS OF (*Cysts, Dermoids*).

**Cyst- or Cysto-**.—In compound words *cyst-* or *cysto-* generally means relating to the bladder (urinary or gall bladder) or to any cyst-like structure.

**Cystadenoma**.—An adenoma containing cysts.

**Cystalgia**.—Pain in the bladder, especially the cases in which there is no recognisable lesion. See BLADDER, INJURIES AND DISEASES (*Chronic Cystitis, Diagnosis*), HEMERIA (*Disorders of the Urinary System, Bladder*).

**Cystatrophy**.—Atrophy of the bladder (injury).

**Cystauchenotomy**.—Incision of the neck (Gr *αὐχὴν*, the neck) of the bladder (injury).

**Cystauxe**.—Hypertrophy of the bladder (injury), especially thickening of the walls (Gr *αὐξή*, enlargement), *cystypertrophia*.

**Cystectomy**.—The operation by which foreign bodies are extracted from the urinary bladder by forceps, the prostatic urethra having been dilated and the membranous urethra divided.

**Cystectomy**.—Excision of the bladder (gall).

**Cystencephalus**.—The teratological type in which the basis cranii is covered by a sac containing fluid and representing the brain.

**Cystic**.—Cyst-like, or bladder-like, or belonging to the urinary bladder or the gall-bladder. See ABDOMEN, INJURIES OF (*Scrupture of Cystic Duct*), NECK, REGION OF (*Cystic Hygroma*), PROSTATE GLAND (*Cystic Disease*), URACHUS (*Cystic Dilatation of*), etc.

**Cysticercus Bovis**.—The scolex or larval stage of the tapeworm, *Tænia mediocanellata*. See PARASITES (*Cestodes, Tænia Saginata*).

**Cysticercus Cellulosæ**.—The scolex or larval stage of the common tapeworm, *Tænia solium*. See HYDATID DISEASE, ORBIT, DISEASES (*Parasitic Cysts*), PARASITES (*Cestodes, Tænia Solium*), TONGUE (*Cysts*), TUMOURS OF THE SKIN (*Cysticercus Cellulosæ Cutis*), VITREOUS HUMOUR, DISEASES (*Parasites*).

**Cysticercus Tenulicollis**.—The larval stage of *Tænia marginata*. See PARASITES (*Cestodes, Tænia Marginata*).

**Cystin**.—A rare deposit in urine (*cystinuria*), consisting of hexagonal crystals, some-

times forming calculi, it is the disulphide of amidopropionic acid ( $C_3H_7NO_2S$ ), it is not soluble in warm water or dilute acetic acid, but is freely so in ammonia, it gives to the urine an odour of sweet linai which soon changes to a disagreeable smell. See PHYSIOLOGY, EXCRETION (*Urine, Sulphur-containing Bodies*), URINE, PATHOLOGICAL CHANGES IN (*Cystinuria, Sediment, Cystin*), URIC ACID, URIC ACID (Auto-Intoxications, *Cystinuria*).

**Cystitis**. See BLADDER, DISEASES OF (*Inflammation, Acute and Chronic*), BLADDER, INJURIES AND DISEASES OF (*Calculi Venere, Discharges*), BLADDER, TUMOURS OF (*Symptoms*), KIDNEY, SURGICAL AFFECTIONS OF (*Pyelitis, Etiology*), PARASITES (*Subacute Combined Degeneration of the Cord, Symptoms*), PELVIS, PERINEUM AND PELVIC FLOOR (*Cystocèle, Complications*), RECTUM, DISEASES OF (*Cystitis a Cause of Rectal Symptoms*), SPERMATORRHOEA (*Cause*), SYRINGOMYELIA (*Cause of Death*), TYPHOID FEVER (*Complications, Cystitis*), URACHUS (*Fistula, Causes of*), URETHRA, DISEASES OF (*Gonorrhea, Complications*), URINE, BACTERIA IN (*Cystitis*).

**Cystocèle**. See BLADDER, INJURIES AND DISEASES OF (*Malpositions, Hernia or Cystocèle*), HERNIA (*Hernia of Special Viscera Bladder*), LABOUR, INJURIES TO THE GENERATIVE ORGANS (*Injuries to the Perineum, Results*), PELVIS, PERINEUM AND PELVIC FLOOR (*Cystocèle*), PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Ventral Complications*), UTERUS, DISPLACEMENTS OF (*Prolapse of the Uterus, Diagnosis*).

**Cystodynia**. See CYSTALGIA.

**Cystoenterocèle**.—A hernia containing both bladder and intestine, when the omentum is also included, it is termed a *cystoenteroepiplocele*, when the omentum and the bladder are the contents, it is named *cystoepiplocele*.

**Cystolithiasis**.—Stone or gravel in the bladder.

**Cystoma**. A cystic growth, e.g. an ovarian cystoma.

**Cystoptosis**.—Projection of the vesical mucous membrane from the urethra due to relaxation of the same.

**Cystopyelitis**.—Inflammation of the bladder and of the pelvis of the kidney. See KIDNEY, SURGICAL AFFECTIONS OF (*Injuries, Results*).

**Cystorrhæxis**.—Rupture of the urinary bladder (e.g. in injuries or in retroversion of the gravid uterus).

**Cystoschisis**.—Extroversion of the urinary bladder or ectopic vesicle.

**Cystoscope, the**. See also BLADDER, INJURIES AND DISEASES OF (*Tumours, Cystoscopic Examination*), GYNÆCOLOGY, DIAGNOSIS IN (*Cysto-*



*scopy*), **HÆMATURIA** (*Differential Diagnosis, Cystoscopy*)—To examine the interior of the bladder visually without a cutting operation is no new idea. In 1805 Bozzini of Frankfurt devised and exhibited an instrument for this purpose, which, however, was found to be quite inadequate because of insufficient illumination. Desmoureaux, in 1854, introduced a cystoscope, which, in 1865, was further elaborated by Dr. Cruise of Dublin. These and all other instruments prior to 1877 may be considered the first stage of the cystoscope, as the light for illumination was *external* to the bladder, but in that year Dr. Max Nitze of Berlin introduced the platinum loop instrument with the light internal, which, although it had various objections, and was soon condemned because of its cumbersome size and defective light, initiated the second stage of the cystoscope or light *internal* idea, and led up to the more perfect instrument of to-day. Not until 1887, however, did surgeons find an instrument worthy of a place in their armamentarium, when, for the first time, the "incandescent lamp" cystoscope was introduced. This instrument has been variously modified, but the Leiter cystoscope, after the model of Mr. Hurry Fenwick, serves excellently for thorough examination of the bladder.

*The Instrument*—Two forms of the cystoscope—the *anterior* and *posterior*--are in use, but in practice the former is alone necessary. It resembles a sound in shape, and consists of the beak, the shaft, and the ocular portion. The beak is hollow, contains the lamp used for illumination, and has a window through which the light is emitted. From the lamp two insulated wires pass along the interior of the shaft to the ocular portion, where they are connected with the electrodes of a battery, the source of electromotive force for light. The shaft is hollow and contains a telescope. At the junction of the shaft and beak, in line with the window in the beak, there is a prism which refracts the rays of light from the object illuminated on to the end of the telescope, and therefore into line with the observer's eye, so that the object is brought into view. On the rim of the ocular portion, upon the same aspect as the window and prism, there is a knob which indicates their position, so that as the instrument is moved to and fro, or turned round within the bladder, we can determine the part of the bladder wall which we examine. When using the instrument it is focussed by withdrawing it from, or approaching it to, the object under examination until the best definition is got. It is necessary to remember that the image is inverted. With the anterior cystoscope the whole surface of the bladder can be seen, but naturally the most difficult portion to bring into view is that immediately around the urethral opening. A suitable battery, provided with a rheostat, is that figured in Schall's catalogue No. 1192.

*To use the Cystoscope*—The most careful aseptic precautions are required, just as in the use of other bladder instrumentation. The cystoscope cannot be boiled without injury, and must therefore be purified by steeping it for a time in carbolic acid or lysol lotion. The other usual precautionary measures to avoid rigors must also be adopted. The pain during examination is no greater than that caused by sounding, and it is only in exceptional cases that anaesthesia is required, but an anesthetic may be exhibited in nervous patients or in those with sensitive bladders. In the female, from motives of decency, an anesthetic may be necessary. In addition to these general measures there are three essentials for examination—

1 The urethra must be of sufficient calibre to easily admit the instrument.

2 The bladder must be capable of holding at least six ounces of fluid.

3 The fluid in the bladder must be transparent.

*Method of Examination*—The urine is drawn off with, by preference, a red rubber catheter. If it be clear—no blood or pus—from 6 to 12 ounces of warm aseptic solution are injected, but if need be the bladder is first washed out that the injected fluid may remain transparent. This should be done as gently as possible to avoid hemorrhage, which might rapidly cause the fluid to be coloured, so that the examination would be difficult, although it is very rarely impossible from this cause. The cystoscope is now introduced, the circuit being *open*, and care taken that the beak is completely within the bladder before it is closed. The circuit having been *closed*, the observer's eye is applied to the ocular portion, and an endeavour made to detect an air bubble, which is almost certainly present at the highest point of the fluid. This is seen as a shimmering globule, and indicates the position of the instrument—gives, so to speak, a landmark from which we can proceed methodically to examine the whole bladder wall. The normal bladder wall has a pale, pinkish-white colour, with small but distinct blood-vessels ramifying on the surface. The pallor varies in degree proportionately to the distension. Bladders vary in their appearance, and it is only after several have been examined that the observer can say whether a departure from the usual appearance is consistent with health or not. During the examination the beak of the instrument must not rest continuously on the wall, as it may become hot and do harm, but this is not apt to occur if ordinary care be taken. If an abnormality be seen it must be closely examined to determine its exact significance. A projection is more readily seen than a superficial lesion. Different meridians are examined by advancing and withdrawing the instrument, but in particular the margin of the *urethral opening*, *neck of bladder*, and *trigone*

are examined. The neck of the bladder is congested in appearance and prominent, but normally smooth in its outlines. The ureteral openings are at each end of the base of the trigone, with, it may be, an inter-ureteral but. They may in some instances be readily seen, but in others require to be closely looked for by judging the distance and proper angle from the urethral orifice. The openings are normally transverse or oblique slits, and may not be detected until the margins are separated by urine coming through them. It is necessary, therefore, to steadily watch for a minute or two the area where we expect to find the opening, so that as the urine is ejected the orifice is seen. If the urine be mixed with blood or pus from the kidney its ejection is more readily observed. Bright blood is readily seen (*v. Case 1, infra*). If the prostate be much enlarged the beak of the cystoscope must be considerably depressed to bring the trigone into view. When the examination is completed the circuit is opened and the instrument withdrawn. It is important to introduce and withdraw the cystoscope with the circuit open, as otherwise the beak, not being surrounded by water, becomes hot, and may burn the urethra. The instrument should not be kept for any time in the air with the circuit closed, as by fusion of the connections it may be injured.

*Value of Cystoscopy.* In disease of genito-urinary conditions, both as a diagnostic and prognostic agent, the cystoscope is valuable. It may enable the surgeon to say definitely that the bladder is or is not the source of hemorrhage, the origin of which cannot be otherwise determined. That is equivalent to deciding in some cases whether the bladder is to be opened or a nephrotomy performed. Further, even if the bladder be the seat of a tumour, cystoscopy may show whether it is suitable or not for removal. Wherever there is doubt as regards the site or nature of disease causing hematuria or pyuria, or when other means have failed to give an accurate diagnosis, cystoscopy should be used. There are two considerations, however, of much importance, which should invariably be borne in mind—*First*, cystoscopy, just as all instrumentation, should as a rule be avoided in vesical tuberculous disease, and, *second*, in patients who are seriously ill, or in whom instrumentation is apt to be followed by aggravation of symptoms, whatever the reason, operation if needed should immediately follow the examination. In those cases of vesical tuberculosis, where the bladder is much inflamed or contracted, cystoscopy should never be resorted to, as in these harm is frequently done, simply, it may be, by over-distension of an inflamed bladder, and the method is brought into disrepute. When, however, there is no obvious tuberculous disease of the prostate, and the bladder is of good capacity, if there be doubt

regarding the source of tubercle bacilli or pus, cystoscopy is valuable to determine whether the kidney is diseased and the bladder healthy. Nephrectomy in primary tuberculous disease of the kidney may be curative, and the decision to perform the operation may depend upon cystoscopic examination showing absence of disease in the bladder. This aspect of the subject can best be appreciated by the consideration of illustrative cases, of which the following may be considered fairly typical—

*Case 1*—Mr J D, *et* 47, complained of pain in the left side and blood in the urine. There was no other symptom, and examination discovered no disease in either the kidney or prostate. Cystoscopic examination showed blood coming from the left ureteral orifice. Nephrotomy was performed, but it was only upon cutting into the kidney that a tumour (sarcoma) was found and nephrectomy carried out. For one year the patient remained in good health, but then a swelling appeared at the seat of the incision, and death resulted one and a half years after operation.

*Case 2*—M S (male), *et* 60. Hematuria only symptom. Cystoscopy showed a pedunculated villous tumour on the right inferior aspect of the bladder about one inch above the right ureteral orifice. The tumour was removed by suprapubic cystotomy. The patient made a good recovery, and there was no sign of recurrence for three years. Now, four years from date of operation, the patient suffers from hematuria, and a large mass can be palpated in relation to the bladder.

*Case 3*—Miss D, *et* 25, chief complaint, frequency of micturition. The urine contains a considerable quantity of pus. Pain in the right loin has been present off and on for four years, and on palpation the kidney is felt to be enlarged. Cystoscopic examination showed a normal bladder. No pus was seen coming from either ureteral orifice. Nephrectomy of the right kidney was carried out. The kidney contained large concretions filled more or less with caseous material.

*Case 4*—J S (male), *et* 50, had symptoms of stone in the bladder, but sounding failed to detect one. Cystoscopy showed a small stone behind a freely movable pedunculated middle lobe of the prostate. An attempt to crush the stone failed as it could not be grasped, and suprapubic cystotomy was had recourse to. This case illustrates that, even when no deep post-prostatic pouch is present, a stone may be missed by the sound. That cystoscopy is of value in cases of post-prostatic pouch for the detection of calculi is undoubted, but the examination and operation should, if possible, be carried out at the same time. In such cases castration is contra-indicated, as the stone or stones must be removed to get rid of the symptoms.

Many cases, differing from the above, might be quoted to emphasise the importance of cystoscopy, if space permitted, but those related will serve to show that assistance in diagnosis may be got by its use in (a) tumours, and (b) calculus of the bladder, and in different affections of the kidney. To detect the presence and nature of foreign bodies the instrument is also valuable. From the point of view of prognosis valuable aid is also got. Thus in tumour of the bladder, if a patient have little bleeding and no pain or frequency of micturition, but suffers from an irremovable tumour, cystotomy should be avoided as a rule. Rest to the bladder by cystotomy may diminish the rapidity of the growth of the tumour, but it may, on the other hand, permit an intrinsic growth to become extensive, so that eventually much greater discomfort and a more rapidly fatal result ensues. Cystoscopy, by showing the extent and attachments of the tumour, may prevent an operation which would be harmful to the patient, while, on the other hand, it may enable the surgeon more strongly to advocate operation, the diagnosis being certain and the hope of cure probable. The latter point is shown in case 2 quoted above, while the following case illustrates the former—

*Case 5*.—Mr. R., æt. 58, three months ago, for the first time, had causeless hæmaturia which lasted for a few days. He had a second attack ten weeks later, and at the present time suffers from a third attack. There is no other symptoms, and external examination is negative. The urine contains many large multi-nucleated cells. Prostate not enlarged on examination per rectum. Cystoscopic examination showed a tumour attached to the prostate. From its extent and position I believed it to be inoperable, and advised no operation.

To decide whether a tumour is operable or not is far from easy, and in some cases impossible, but in other cases there is not much difficulty. If in doubt I think the patient should have a cystotomy. The cystoscopic examination must be very thorough, and should preferably be carried out with different degrees of distension of the bladder. A tumour in an imperfectly distended bladder may appear as a mass, while if the bladder be more fully distended the growth may be seen to be made up of several separate masses affecting a much larger area of the viscus. I have seen this in two cases recently, in one of which cystotomy showed the disease was inoperable, while from cystoscopy I believed the disease could be eradicated.

The subject of cystoscopy has in the last ten years been much widened by the introduction of apparatus to treat conditions per urethram, or to catheterise the ureters. Both of these procedures I believe to be of little practical importance in the male, and sometimes, indeed, dis-

tinctly injurious. In the female, however, the method introduced by Dr. Kelly, of Baltimore, is more valuable, although short of metrical catheterisation, equally good results can in the majority of cases be got by the anterior internal light cystoscope without the disadvantage of urethral dilatation incident to the use of Kelly's cystoscope. Examination by Dr. Kelly's instrument is simple, and metrical catheterisation with it can be rapidly executed. A complete account of the instruments required for examination and the technique is contained in Kelly's work *Operative Gynecology*. The fundamental principles of examination are—

- 1 The introduction of a simple cylindrical speculum.

- 2 The atmospheric distension of the bladder by posture.

- 3 The illumination and inspection of the bladder by a direct light.

The instruments required are: A strong light, a head mirror, vesical specula with obturators, a urethral dilator and dilator, an evacuator for removing urine, and a metrical searcher.

Local anesthesia may suffice, and undue dilatation of the urethra is unnecessary.

**Cystotomy.**—The operation of opening into the urinary bladder (for the removal of a calculus or tumour), either by the perineal route (*median, lateral, or bilateral*), by the abdominal (*suprapubic*), by the rectal, or by the vaginal route.

**Cytisine.**—An alkaloid ( $C_{10}H_{11}N_2O$ ), also named *adonine*, found in laburnum seeds (*Cytisus laburnum*), and acting as a poison.

**Cyto.**—In compound words *cyto-* (*κίτος*, a cell or pot) means relating to a cell (*Cytoblast*, for instance, is the cell nucleus, *cytote* is a cell without a nucleus, *cytodivision* is cell-division, *cytogenesis* is cell-formation, *cytology* is the science of cells, *cytometria* is the reticulum of the protoplasm of cells, *cytoplasm* is the protoplasm of the cell as distinguished from that of the nucleus (*cytoplast*), *cytotox* are parasites of cells (e.g. protozoans, such as coccidia).

**Cytodiagnosis.**—One of the most recent advances in clinical pathology consists in the histological examination of various fluids derived from the patient, whereby their cell content may be qualitatively and quantitatively estimated. Certain secretions which have been altered by disease, and inflammatory and passive exudates, are found to contain cellular elements which vary with the cause of the disease process and with the stage at which this has arrived. It has, of course, long been the custom of careful observers to search various secretions and exudates for histological evidences of new growth where the presence of this has been suspected. In pathological urines, too, the various cellular

elements of the deposit have long been examined critically for purposes of diagnosis, and the localisation of lesions of the urinary tract has been assisted by recognition of the kinds of epithelial cell present, together with its reaction to certain dyes, notably alizarin blue. Again, the character of the cells present in the sputum has been microscopically investigated, with results bearing upon the nature and situation of pulmonary lesions: the presence of large numbers of eosinophile leucocytes in asthmatic sputum may be instance. The contents of the blebs in certain bullous skin eruptions have been similarly dealt with: here also the existence of eosinophiles in relatively large numbers in cases of true pemphigus has been noted. Lastly, most pathologists have recognised the advisability of making a cytological as well as a bacteriological examination of pus obtained from any source. All these are instances of cytodagnosis. But the method has taken a new departure during the past few years, and various inflammatory exudates have been systematically examined with a view to differentiating, by means of their cell content, the causes of the disease. It is to this investigation particularly that the term cytodagnosis is now applied.

In this field the chief work has been done by the French school of clinical pathologists, led by Vidal, Sicard, and Ravaut. These observers have obtained results which justify them in deducing certain formulæ of considerable diagnostic and prognostic value. These formulæ are receiving daily confirmation and qualification by other investigators, including several English microscopists. The technique of the method of investigation is exceedingly simple. The fluid to be examined is collected, films are made either directly or after centrifugalisation—depending upon the richness of the material in cells and whether a quantitative examination is, or is not, needed,—and these films are examined fresh and after appropriate staining. If the fluid clots readily, as in the case of most pleural effusions of inflammatory origin, the clot is broken up prior to examination. The various cells present are then noted, and a differential count is made after the manner of a white blood cell count. The cells met with are of four types. Three of these are identical with the commonest cells present in blood—the red blood corpuscle, the polymorphous leucocyte, and the lymphocyte. The fourth cell is the endothelial plate derived from the lining membrane of the particular cavity whence the fluid has been obtained. Consideration of the significance of hæmorrhagic effusions lies outside the scope of this article. The endothelial cell varies in its individual characters according to the situation investigated, but also, and to a larger extent, according to the nature of the pathological process present. Thus it is smaller and shows

signs of greater activity in fluids which result from inflammations, especially when acute, than in those which result from passive exudations (transudations). But it is to the relation in numbers existing between the two forms of leucocytes that attention has been specially directed.

In the case of *pleural effusions*, and in the case of fluid removed from the *meninges* by lumbar puncture—the two investigations where the method proves to be most profitable—the following formula has been sufficiently established. *A high lymphocyte count during the early stages of an inflammatory process indicates that the cause is a tuberculous infection, a high polymorphous count during the same stages indicates that the cause is an infection by some "pyogenic" organism—streptococcus, staphylococcus, pneumococcus, meningococcus, gonococcus, colon bacillus, etc.* The qualification as to the stage of the disease is necessary, because it has lately been shown that during the stage of convalescence from "septic" infections, or after these infections have become chronic, the polymorphous cells present in the exudate may give place to lymphocytes. It follows, therefore, that a change in nature of the cells from the polymorphous variety to the lymphocyte indicates a good prognosis so far as the stage of infection is concerned. This change is not infrequently seen when cytodagnostic observations are made in cases of meningococcal meningitis, in which recovery, at least from the infective stage, is not uncommon. With regard to the actual percentage of the dominant cell present no definite statement can be made, the figures, however, are usually sufficiently pronounced in the one direction to leave no difficulty in applying the formula—75 per cent, more or less, is a figure very commonly obtained. The figure may be much higher the writer has counted a first 100 cells in the effusion from a case of primary tuberculous pleurisy, without coming across a single polymorphous cell. The occurrence of a pyogenic infection together with a tuberculous infection shows itself by the presence of a higher polymorphous count than in a tuberculous infection alone. Indeed, in actual practice, the tuberculous part of the disease in these cases is not suggested by the cytodagnostic method. Thus, pleural effusions occurring in the course of pulmonary phthisis obey the "septic" part of the formula, as also does a terminal streptococcal meningitis in the course of a tuberculous meningitis.

In the case of *ascitic fluids* the formula is by no means so trustworthy, however, the absence of microbic infection may be inferred from the presence of passive endothelial cells and the comparative absence of polymorphous cells and lymphocytes. In the case of fluid from inflamed *joints* there is as yet no sufficient body of evidence at hand to warrant any conclusions.

It will be noticed that the formula given above deals only with *qualitative* results of cell counts. Some important indications follow the *quantitative* examination of certain fluids with regard to their cell content, and this is particularly so with the cerebro-spinal fluid. Normal cerebro-spinal fluid is free from cells, or contains an occasional lymphocyte only. A condition of lymphocytosis exists in certain diseases of the central nervous system, and appears to be proportional to the degree of meningeal involvement present. Thus, a slight lymphocytosis is found to occur in *tuberculous* and in *insular sclerosis*, a somewhat higher cell count is present in general paralysis of the insane, in cerebral syphilis, and in the case of cerebral tumours involving the meninges. It may readily be seen that these facts—always to be taken in connection with the more immediately clinical aspects of the case—provide considerable assistance in differential diagnosis of nervous diseases, and form an additional reason, if this were necessary, for the more extended use of lumbar puncture as a means of clinical examination in obscure diseases of the nervous system. It need scarcely be pointed out that the cytodagnostic method may yield valuable information in deciding between organic and functional nervous diseases.

**Cytolysins.**—Bodies causing dissolution of cells.

**Cytoryctes Variolæ.**—Protozoon-like bodies found by Guarnieri in the skin lesions of smallpox, they may be the cause of the disease; they are found also in vaccinia (*Cytoryctes Variolæ*).

**Cytotoxines.**—Poisons produced by cells and capable of destroying cells.

**Cytozoa.** Cell-parasites, sporozoa, such as the *Coccidium oviforme* found inside the epithelial cells which line the bile ducts of the rabbit's liver. See PSOROSPERMIAS.

**Dabola.**—A poisonous snake, one of the Viperidae (or true vipers). "Russell's viper" of India and Ceylon. See SNAKE-BITES AND POISONOUS FISHES.

**Dacry.**—In compound words *dacry* (from *ἡ δάκρυα*, a tear) generally means relating to the lacrimal gland or duct or (rarely) to the phenomenon of weeping, thus *dacryadenitis* is inflammation of the lacrimal gland, *dacryadenitis* is pain in the lacrimal gland, and *dacrygelonitis* (*ἡ δάκρυα*, a tear, and *γελῶ*, I laugh) is a form of insanity characterised by alternate fits of excessive weeping and laughing. *Vide infra*.

**Dacryoadenitis.**—Inflammation of the lacrimal gland, also spelled *dacryadenitis*.

**Dacryoblenorrhœa.**—Discharge (mucous) taking place from the lacrimal sac and ducts.

**Dacryocystitis.**—Inflammation of the lacrimal sac or gland, catarrhal or suppurative. See LACRYMAL APPARATUS, DISEASES OF (*Gland, Inflammation*). The occurrence of a mucous discharge from the sac is termed *dacryocystoblenorrhœa*, prolapse of the sac is *dacryocystoptosis*, while a hernia of it is *dacryocystocele*.

**Dacryolith.**—A calculus or chalky concretion blocking a duct of the lacrimal gland, or a canaliculus, of the sac. See LACRYMAL APPARATUS, DISEASES OF (*Diseases of Gland*).

**Dacryoma.**—Obstruction of one or both of the puncta lacrymalia, preventing the passage of tears into the lacrimal sac.

**Dacryon.**—The point at the side of the root of the nose where the frontal, the lacrimal bone, and the ascending process of the superior maxilla meet, used as a landmark in craniometry.

**Dacryops.**—A cyst-like distension of a duct of the lacrimal gland, also a "watery eye." See LACRYMAL APPARATUS, DISEASES OF (*Gland, Diseases, Cystic Growth*).

**Dacryosolenitis.**—Inflammation of the lacrimal duct (from *ἡ δάκρυα*, a tear, and *σολήνη*, a channel).

**Dacryosyrinx.**—A fistula lacrymalis (*ἡ δάκρυα*, a pipe).

**Dactylitis.**—Inflammation of the fingers or toes (*ἡ δάκτυλος*, a finger or toe), especially that form in which there is marked enlargement of the digits of a syphilitic (gummatous) nature (*dactylitis syphilitica*). See BONE, DISEASES OF (*Tuberculous Disease in the Interior of Bones, Tuberculous Dactylitis*); BONE, DISEASES OF (*Syphilitic Dactylitis*); FINGERS (*Diseases of Bones and Joints, Dactylitis*); HAND (*Bones, Dactylitis*); SYPHILIS (*Tertiary, Bones and Joints*).

**Dactylolysis.** Spontaneous amputation of the fingers or toes met with at birth, and ascribed by some to the pressure of the umbilical cord or of bands of amnion or of lymph, and by others to a cutaneous lesion (*viz* proliferation and downgrowth of the surface epithelium). It is in connection with the last-named theory that the term *epithelial dactylolysis* has been given (Menzel), perhaps this congenital state is allied to the disease called anhidrosis (*q.v.* vol. 1 p. 76).

**Dactylotheca.**—A finger-cot, a protective covering used in certain examinations and manipulations.

**Dæmonomania.**—A form of insanity in which the patient (usually suffering from religious melancholia) imagines himself the subject of possession by devils, demonomania.

**Dairies.** See MILK (*Industrial, Hygiene of the Dairy*), COW-SHEEDS.—Both in English and Scots sanitary law dairy means "any farm, farm house, cow-shed, milk-store, milk-shop, or other place from which milk is supplied, or in which milk is kept for purposes of sale."

**Dalby's Carminative.**—A soothing preparation containing about two and a half minims of laudanum to the fluid ounce. See TOXICOLOGY (*Alkaloids and Vegetable Poisons, Opium and Morphine*).

**Daltonism.**—Colour-blindness, so called because John Dalton, chemist, studied and carefully described the condition of red-blindness or anerythropsia in himself. See COLOUR VISION (*Colour-Blindness in Achromatopsia*).

**Damiana.**—A Mexican plant (*Turnera aphrodiasiac* [?] or *Baylonia veneta* [?]), regarded as possessing aphrodisiac qualities and as being a nerve tonic.

**Damp.** See STOMACH AND DUODENUM, DISEASES OF (*General Etiology, Cold and Damp*).

**Dance, St. Vitus's.** See CHOREA.

**Dancer's Cramp.**—Cramp of the calf muscles, especially occurring in ballet-dancers.

**Dancing Mania.**—A form of epidemic mania (met with in the Middle Ages, often in association with religious excitement) in which dancing and gesticulating were prominent symptoms, St. Guy's or St. John's dance, pandemic chorea, choromania.

**Dandelion.** See TARAXACI RADIX.

**Dandriff or Dandruff.**—Scurf or dead scurf-skin still adhering to the skin or to hair on the skin, pityriasis. See ECZEMA (*Mycosiform, Pityriasis Capitis*).

**Dandy Fever.** See DENGUE.

**Daneverd.** See BALNEOLOGY (*Sweden*).

**Dangerous Trades.** See ANTHRAX, TOXICOLOGY, TRADES, DANGEROUS.

**Daphne Mezereum.**—The root of this plant yields the mezereum of the United States Pharmacopœia, cases of poisoning from eating the berries have occurred, the bark is official in the British Pharmacopœia, and contains *daphnin*, a crystalline glucoside ( $C_{15}H_{16}O_6$ ), which boiled with dilute acids gives *daphnetin* ( $C_9H_8O_4$ ) and glucose. See MEZEREI CORTEX.

**Darier's Disease.**—A rare chronic

disease of the skin, characterised by the occurrence of papules in regions of the body well supplied with sebaceous and sudoriferous glands (e.g. the axillæ and the groins), by the later formation of greyish-brown or black crusts, and by the still later development of inflammatory nodules and ulcers, the disease shows some resemblance to lichen planus, it is practically an incurable malady, but good can be done by the use of antiseptic remedies and thorough cleansing, which may prevent infection of the affected areas of skin with pyogenic bacteria.

**Darjiling.** See BALNEOLOGY (*India, Sulphur Waters*).

**Dartos.**—The layer of subcutaneous tissue in the scrotum containing much unstriated muscle, and forming a sac for each testicle, it contracts under the influence of cold or of irritation. The name is derived from the *Gr.* *daprós*, excoriated.

**Dartre.**—A generic name applied somewhat vaguely to various skin diseases, including especially herpes, pityriasis, and lichen, which were supposed to be all due to the same constitutional state.

**Darwinianism or Darwinism.**—Charles Darwin's theory of the origin of species, etc., in which evolution by natural selection and survival of the fittest is the central assumption. See HEREDITY. *Darwin's tubercle* is the projection on the intumed margin of the helix of the external ear in the human subject supposed to represent the tip of the ear of apes and other lower mammals.

**Dasytes.**—Hairiness (from *Gr.* *dasís*, hairy), especially general congenital hairiness (hypertrichosis congenita), individuals so affected have been termed "hairy men," "missing links" of the Darwinian theory, "Esau," and "homines pilosi", pilosism, polytrichia, hirsuties.

**Date of Delivery.** See FORTUS AND OVUM, DEVELOPMENT OF (*Mature Fœtus*), PREGNANCY, DIAGNOSIS (*Probable Date of Confinement*).

**Daturæ Folia.**—The dried leaves of *Datura fastuosa* and *Datura Metel*, they have a bitter taste, a peculiar odour, and the same action as stramonium leaves (*qv*), they are official in India and the Colonies, being described in the Indian and Colonial Addendum (1900) to the British Pharmacopœia (1898).

**Daturæ Semina.**—The dried seeds of *Datura fastuosa*, they are described in the Indian and Colonial Addendum (1900) to the British Pharmacopœia (1898), they have the same action as stramonium seeds (*qv*), and there is a *Tinctura Daturæ Seminum*, given in doses of 5 to 15 m.

**Datura Stramonium.** See STRAMONII FOLIA, STRAMONII SEMINA

**Daturine.**—An alkaloid obtained from *Datura Stramonium*, etc., identical with hyoscyamine and isomeric with atropine. See ALKALOIDS (*Vegetable*), STRAMONII FOLIA, etc.

**Dauglish's Bread.**—Patent aviated bread made from flour by an admixture with carbonic acid water under pressure by means of special machinery

**Davainea Madagascariensis.**—A rare cestode, found in Madagascar, Mauritius, and Bangkok. See PARASITES (*Cestodes*)

**Davos Platz.** See THERAPEUTICS, HEALTH RESORTS (*Sunt island*)

**Dax.** See BALNEOLOGY (*France, Thermal Waters*)

**Day Blindness.**—Inability to see (either at all or without pain) in the daylight, hemeralopia

**Daymare.**—A condition of extreme terror or distress, resembling that seen in cases of "night terrors," occurring in an individual in the waking state, allied to epilepsy. See NIGHT TERRORS (*Nightmare*), RHEUMATISM IN CHILDREN (*Nervous Affections, Day Terrors*)

## Deafmutism.

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See also MENINGITIS, TUBERCULOUS AND POSTERIOR BASIC (*Prognosis*)

By dumbness we mean the want of power to articulate sounds. All classifications of cases of dumbness are open to objection. The following two great divisions require special discussion—

1 Deafmutism or deafdumbness. Dumbness due to deafness

2 Dumbness associated with idiocy or dementia

In addition to these, dumbness may be the result of damage to the brain, *e.g.* aphasia, where there is sometimes entire loss of speech, due to a lesion in the third left-frontal convolution, apoplexy, bleeding on or into the brain, embolism of a cerebral artery, tumour of brain, etc.

Further, dumbness may be part of a more general paralysis, such as lead paralysis, or it may be due to pressure on the hypoglossal nerve. Dumbness may also be a feature of hysteria

**Deafmutism.**—The deaf and dumb are not otherwise physically peculiar. Placed under

similar conditions they develop equally with hearing children. At six or seven, when the deaf child enters school, he is like the hearing child of two or three years, and it is a curious fact that his head is half an inch less in circumference than the hearing child of the same age. Such is the influence of hearing on the development of brain

All deaf mutes are not stone deaf. Most hear loud noises or shouting, some distinguish vowels and consonants, and I have had them beat time accurately to music of all kinds when the sounds of the phonograph were conveyed to their ears by the tubes belonging to the instrument, although their eyes were covered carefully to prevent their seeing the manipulations about the instrument. But all deaf mutes are so deaf that they cannot hear ordinary talk and cannot be taught with hearing children. Speech is normally the result of hearing, and when there is little or no hearing the child will not speak. Further, if at four, five, or six years he lose his hearing, his speech will leave him

*Deafmutism is either congenital or acquired*—

In Britain about 50 per cent of the cases belong to each of these two great classes. But in some parts of America and the continent of Europe, epidemic cerebral spinal meningitis raises the proportion of acquired cases, which with reference to the congenital cases bear a ratio of nearly two to one. Still, although epidemic meningitis is not common in Britain, diseases of the head, of which meningitis is the chief, are responsible for a large number, next to meningitis as causes of acquired deafness comes scarlet fever, and after this disease measles. These three diseases account for nearly 60 per cent of our cases of acquired deafness. Now, when these diseases take away hearing, they often take it all away, and, further, they often damage the intelligence of the child in other ways, hence come these two facts—

1 When testing the hearing of the deaf and dumb it is common to find a congenitally deaf child with more hearing than a child who has lost his hearing after birth

2 Teachers often find the congenitally deaf child bright and clever, and the acquired deaf mute slow and stupid

Congenital deafness, as we have seen, accounts for about half the number of cases of deafmutism in Britain. Many cases supposed to be due to disease in early life are really cases of congenital deafness. This mistake is due partly to the fact that parents are slow to admit the presence of a family defect, and partly to the fact that diseases occurring in very early life are apt to be credited with whatever defect is discovered later. Thus falls, frights, etc., are often credited with having caused deafness which has existed from birth

In every institution examples may be found of deaf-mute children who have one or two deaf

parents or grandparents, and of two or more deaf-mute children belonging to one family. Statistics from British institutions have been fully dealt with in the author's chapter on congenital deafness, "Deafmutism, a Clinical and Pathological Study," but it is interesting to examine statistics so entirely divorced from European influence as those of Japan must be. In the *Report of the Tokio Institution* for 1896, Mi Shimpachi Koushi, director of the school, says, "Out of sixty-six dumb pupils in this school, there is one pupil whose grandfather was deaf, one whose grandfather's brother was deaf, and one whose father's grandfather was deaf. The number of pupils who inherited dumbness directly from their parents is only three, whilst fourteen dumb pupils were born of parents who married their cousins." Now these statistics are quite parallel with similar ones from British and American institutions. Then take the following instances from the Glasgow records—

1 In the F family there are 10 children—5 deaf born, and 5 hearing. The parents hear.

2 In the Ayrshire family there have occurred during the present century 42 cases of congenital deafness, deaf-mute children are still being born into this family, and one is being educated in the Glasgow institution now. There spring from these statistics, for the fuller consideration of which the author's work may be consulted, these two conclusions—

1 The congenitally deaf and those related to them should not marry.

2 Intermarriage of blood relations should be strongly discouraged.

*The first position*—that the congenitally deaf should not marry—is generally conceded by those who work amongst the deaf, but the present arrangements for the education of the deaf, and their management in missions and institutes for the deaf during the period of adolescence, is eminently fitted to encourage union between the congenitally deaf. If not during the school period, at least during the period of adolescence, everything should be done to discourage the association of the deaf and dumb with each other, and the danger of their meeting with those similarly afflicted should be constantly kept before the congenitally deaf by those in charge of them. There are a few bad families, to the members of which it would be well to prohibit marriage altogether. Here is an example. It is taken from the *Sheffield Evening Telegraph* for 26th June 1896—"At an inquest yesterday on William Earnshaw, fifty-nine, a St Pancras saddler, it was stated that the relatives could not identify the body, as the wife and sister were blind, deaf, and dumb, and that the four children were deaf and dumb. The deceased was deaf and dumb, and was so when he was married." Now such families as this are responsible for a large number of the deaf mutes

in our institutions. I do not think it would be wrong to prohibit marriage to the members of such families. I do not say that all the congenitally deaf should be forbidden marriage, but where the defect is so pronounced that the result is a foregone conclusion, I think the interests of the State should go before those of the individual.

*The second position*—that blood relations should not intermarry—can hardly be dealt with by legislation, and if the facts were known there should be no need for legislation. On the other hand, prohibition involves no injury to any one. The facts are these. The intermarriage of consins will emphasise in the offspring whatever defects are characteristic of the family, there is no chance of these being neutralised or dying out as the result of the marriage, as is usually the case when persons previously unrelated get married. Given perfectly healthy consins with no tendency to any disease, intermarriage would not do harm to the offspring, and might conceivably do good. But as such consins do not exist, marriage between consins is often disastrous and is always imprudent.

*THE MORBID ANATOMY OF DEAFMUTISM*—It is curious that malformation of the external ear is seldom found in deafmutism which so often is congenital. Probably not even in one per cent of deaf-mute children does such a condition exist. In the middle ear pathological conditions are much more common, but these consist of the usual changes—perforations of the membrane, loss of the ossicles, necrosis of the petrous portion, etc. But, of course, it is not to these that the deafness is due. The acquired deafmutism is almost always due to disease of the internal ear which has spread from the middle ear, and caused inflammatory changes involving destruction of the membranous labyrinth and of the nerve structures which it supports. In a number of cases the danger appears to approach from the side of the brain—meningitis,—and more rarely still, the damage to the inner ear is due to a primary labyrinthitis.

In congenital deafness the changes on which the deafness depends are not essentially different from those described above, except in cases which depend on arrest of development or malformation. They consist of obliteration of the normal nervous structures by inflammatory new formations, chiefly ossous. It is not possible to estimate the proportion of cases due to malformation, but it is undoubtedly smaller than was formerly supposed. After a series of years it cannot in many cases be decided whether a structure has been obliterated or has not been developed.

*THE TESTING OF DEAF MUTES*—It will be found impossible to apply reliable and accurate tests to very young deaf mutes. The presence or



absence of hearing may be determined, but the amount of it cannot be ascertained till the deaf child has been so far educated as to understand the nature of the tests. Hence it is not worth while testing deaf children till they have been a year at school. In the interval, of course, the teacher has discovered any cases in which much hearing remains. The best tests are a dinner-bell so arranged that one note may be struck at a time, and the human voice used in the production of vowels and consonants. Bezold<sup>1</sup> of Munich used the whole gamut of the musical scale, and made a very interesting discovery, viz that in some cases islands of hearing exist in the surrounding ocean of silence or deafness. Those islands, of course, represent the less injured pieces of the cochlea, and sometimes include only a note or two, sometimes a half or a whole octave. These observations of Bezold give a very strong support to the Helmholtzian theory of the function of the cochlea, but as the islands seldom coincide with the tones of the human voice, and as the continued tones are seldom very distinctly heard, they will probably be of little use in the practical training of the deaf.

**THE DISTRIBUTION OF DEAFMUTISM**—The proportion of the deaf to the hearing varies in different countries, but it is governed by causes which operate alike in all countries. The following table gives a review of the deaf-mutism in various countries. It gives the number of deaf mutes per million living as well as the ordinary rate—

	Rate per million	
Switzerland	2452 or 1 in	408
Austria	1307	765
Hungary	1263	792
Sweden	1023	977
Prussia	1019	981
Finland	1018	981
Canada	997	1003
Norway	950	1052
Germany (without Prussia)	931	1074
Portugal	750	1333
Ireland	715	1398
India	685	1459
United States	659	1514
Denmark	650	1538
Greece	646	1548
France	626	1600
Italy	537	1862
Scotland	530	1885
Cape Colony	525	1904
England	489	2043
Spain	459	2178
Belgium	445	2247
Australasia	371	2692
Holland	335	2985
Ceylon	231	4328

The causes which determine these variations

<sup>1</sup> *Das Hörvermögen der Taubstummen*, 1896

in the deaf-mute rate are various. Speaking generally, in prosperous flat countries like England, where the population is well fed and housed, where infectious diseases are carefully treated, and where communication is rapid, the deaf-mute rate is moderate. On the other hand, in a mountainous country like Switzerland, sparseness and stasis of the population make for increase of consanguineous union and an increase of congenital deafness, while the greater difficulty of combating the ravages of the exanthemata makes for an increase of acquired deafness, and the deaf-mute rate tends to be high. Similarly new colonies like Australia have a lower deaf-mute rate than the old colonies like Canada, consanguineous union being less common.

**DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DEAFMUTISM**—When a dumb child is brought to the surgeon, the first thing he should do is to test his hearing. The child is, say, three years old, and his mother is dissatisfied with his speech. She does not think him dumb. He says Ma-ma and Pa-pa, and the mother thinks this proof that he hears. Further, she has noticed that loud sounds, such as the slamming of a door or the fall of a shovel, startle him. Now the facts are that the child says Ma-ma by imitation, he lip-reads, and he turns at the loud noise, either because he hears nothing but very loud sounds, or because he feels the vibration caused along the floor or wall when the door is slammed. It is generally easy to settle the question of the presence or absence of hearing.

Engage the attention of the child with your watch or knife, and let an assistant slip unnoticed behind the child's back with a gong or a bell, and while it is being sounded watch the child's face. If he hear he will turn, or wince, or wink, or in some way show by his expression that he has been affected by the sound. If he show nothing of this kind he will be too deaf for teaching in the schools for hearing children. The more accurate testing of deaf mutes is interesting and has been referred to, but this test is enough for the child as he is first brought before the surgeon. The prognosis in such cases is almost uniformly bad as far as the hearing is concerned. It is best to tell the mother that her child will be a deaf mute, and will require to be educated by special methods. In the meantime she may be made to look upon the development of hearing as a possibility, and she should be encouraged to ply the ears of her child with all sorts of sounds, especially with sounds of all pitches, e.g. the notes of the violin, piano, concertina, etc., with the view of encouraging such development. But this is rare. I have seen such growth of hearing in one instance, or rather in two children in one family, but it is not common enough to set before the parents of deaf children as a probability. Apart from the deafness, the prognosis in older chil-

dren should be based on the evidence of natural ability shown by the child. Congenitally stone-deaf children often make bright pupils. Semi-deaf children who have had their hearing damaged by meningitis and scarlet fever are often dull and make little progress.

The treatment of deafmutism divides itself into surgical and educational. Five to ten per cent of deaf-mute children have chronic suppurative disease of the middle ear with or without necrosis of the ossicles or internal tympanic wall when they are sent to school. These cases require treatment on ordinary surgical lines. But a far larger number of deaf-mute children have enlarged tonsils and post-nasal adenoids. Now the removal of these growths will not make the children hear, but if any serious attempt is to be made to make the children speak well, these growths should be removed wherever they are well marked. As deafmutism depends in nearly every case on destruction of or absence of the internal ear, efforts having for their object the restoration of hearing will necessarily fail. But the question arises whether an attempt should be made to use the partial hearing which so many deaf children have. Now this, like almost all questions connected with the education of the deaf, must be decided with reference to the circumstances and position of the child. If the parents can afford it, the child should have a teacher all to himself, and with the help of conversation tubes, phonograph, etc., everything should be done to teach the child through his auditory nerve. But in a schoolroom, with classes of six to twelve in number, this is clearly impossible. Further, teaching by conversation tube is resented by teachers; they do not like the shouting involved. A few teachers in this country and in America have tried this *acoustic method*, and find that the speech of oral pupils is improved in about 15 or 20 per cent of the cases by this method of teaching. If carefully and persistently used this method may be made a great help to oral pupils. Seldom will it be possible to teach the deaf-mute child by the auditory nerve alone.

On the general question of educational methods it is not the intention of this paper to dilate at length, but it may be stated that in the opinion of the writer the *oral method* should be adopted for the preservation and perfecting of the speech which the semi-mute have still left. Also when there is any great remnant of hearing the oral should be combined with the acoustic method, and in these two classes of cases the semi-mute and the semi-deaf signs should be excluded as much as possible. But there remains the great class of the true deaf mutes—those who have never spoken and who have no hearing which can be used. These form two-thirds or three-quarters of the entire number of the deaf and dumb. When one of these children can be

taught in private and have the entire attention of a teacher, the oral method will generally give results gratifying to its parents and of great benefit to the child, and it should only be given up for the finger method if fair progress is not being made with the education of the child. In an institution many children, perhaps most, will do better on the finger method or on a combined method. For the first year the writer thinks all deaf-mute children should be educated on the oral method. At the end of this period the teacher will know if the child is likely to make a good oral pupil, and will regulate his future accordingly. This involves two schools—an oral and a finger school—in every large centre of population, from the former of which all signs are as far as possible excluded. Like the hearing child the deaf mute should be sent early to school, and in this country both law and expediency have fixed seven years as the best time for beginning education at school. For Scotland in 1891 and for England in 1894 an Act came into force, the chief provisions of which are these—Education is compulsory from the age of seven to that of sixteen, and the duty of seeing that this is carried out is laid on School Boards or the school authorities of the parish where the parents of the deaf children reside. The authorities, in carrying out the Act, are at liberty to provide a school of their own, or they may send the child to a certified school or institution outside their bounds, the parents being allowed a reasonable amount of liberty in the choice of a school. Where the parents from poverty are unable to pay the fees, the school authority is required to pay, not only for the education, but also for the maintenance of the child.

#### LIST OF TRADES, ETC., IN WHICH THE DEAF AND DUMB ARE EMPLOYED

(Report of the Glasgow Mission to the Deaf and Dumb, 1894-95)

Artist	1	Brought forward	83
Art Metal Workers	2	Dressmaker	2
Bakers	4	Domestic Servants	3
Beltmaker	1	Draughtsman	6
Blacksmiths	2	Dressmakers	10
Boilermakers	6	Dyer	1
Bookbinders	20	Engine Fitters	6
Bookfolders	4	Fancy Boxmakers	6
Boxmakers	5	Fishing Tackle Makers	2
Brass Engravers	7	Gardeners	2
Brass Finisher	1	Glass Decorators	3
Bricklayers	2	Glass Stainers	2
Brickmakers	2	House Joiners	3
Brushmakers	4	Iron Workers	3
Butcher	1	Jewel Case Makers	2
Cabinetmakers	2	Jeweller	1
Calenderer	1	Labourers	16
Carpenter	1	Lamplighters	2
Carpet Designers	5	Lithographic Artists	15
Caulkers	3	Marble Cutters	2
Chairmaker	1	Mill Workers	4
Clerk	1	Moulders	3
Compositors	5	Needlewomen	8
Confectionery Worker	1	Painters	2
Cooper	1	Patternmakers	4
Carry forward	83	Carry forward	190

Brought forward	190	Brought forward	225
Purse-makers	2	Tinsmiths	6
Riveters	1	Umbrella Makers	5
Saddler	1	Upholsterers	3
Sculptor	1	Washerwomen	3
Ship Joiners	1	Weavers	5
Shoemakers	6	Wood Carvers	1
Silver Engraver	1	Wood Engravers	1
Tailors	16	Wood Turner	1
Ticket Writers	2		
		Total	253
Carry forward	225		

**DUMBNESS FROM IDIOCY.**—Dumbness is a necessary result of great deafness happening early in life, and of congenital deafness, but dumbness is no necessary concomitant or result of idiocy. Most imbecile children can be taught to speak. Dr Ireland says: "The lower classes of idiots never learn to speak at all. Out of 103 cases of which I have notes 36 were found mute on entry, and 67 could speak more or less. The average time at which they began to speak was 4 years and 3 months. Only four were noted as having spoken at one year." Children of this class, when they are dumb, are so because they have no ideas to express, and speech develops as education proceeds. At Larchmont, Stirlingshire, Mr Skene, the superintendent of the institution there, showed the writer a cretinous idiot who had not only improved in growth and in general condition very remarkably, but whose speech showed an equally satisfactory development under the use of thyroid gland and extract, and this treatment promises no less brilliant results when applied to cretinous children than it has given in myxœdema. Two classes of idiotic children remain to be mentioned—the deaf-mute idiot and the aphasic idiot. Both of these should be educated as idiots, and not in schools for the deaf and dumb. The aphasic idiot or the idiotic aphasic hears perfectly, but is not intelligent, and does not do well in the schools for the deaf and dumb. The deaf-mute idiot is hardly capable of education in the ordinary sense at all. Most idiotic children have good musical ears, and perform and sing in action songs very well.

**Deafness.** See ALCOHOLISM (*Intoxication, Dulness of Hearing*), AUDITORY NERVE AND LABYRINTH (*Physiology, Test, Nerve Deafness, etc.*), BRAIN, TUMOURS OF (*Localising Symptoms, Hearing*), BRAIN, CEREBELLUM, AFFECTIONS OF (*Tumour, Auditory Nerve*), CINCAGRIC INFLAMMATION (*Clinical Forms, Deafness*), CRÉTINISM (*Causation*), EAR, EXAMINATION OF (*Simulated Deafness*), HEREDITY (*Inheritance of Disease, Deafness*), HYPNOTISM (*Experimental Phenomena, Deafness*), HYPNOTISM (*Therapeutic Uses, Deafness*), MENINGITIS, TUBERCULOSIS AND POSTERIOR BASIL (*Prognosis*), MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Symptoms, Ear*), NOSE, POST-NASAL ADENOID GROWTHS (*Symptoms*), PHYSIOLOGY, THE SENSES (*Hearing*), RHEUMATISM, CHRONIC (*Clinical Features*), SPINE, SURGICAL AFFECTIONS (*Causation, Disease*), SYPHILIS (*Children,*

*Ear*), THYROID GLAND, MEDICAL (*Myxœdema, Special Senses*), TONSILS, DISEASES OF (*Enlarged Tonsils, Symptoms*).

**Death.** See ABDOMEN, INJURIES OF (*Causes of Death*), ADOLESCENT INSANITY (*Nerve Breakdown and Mortality Statistics*), ANÆSTHESIA (*Deaths under Ether, under Chloroform*), ANÆSTHESIA, COMBINED ANÆSTHETICS (*A C E. Mixture, Deaths under*), ASPHYXIA (*Causes*), BRAIN, TUMOURS OF (*Prognosis*), BREATH (*Determination of Death*), CLIMATE (*Death-Rate in Cold Climates*), DEATH, SIGNS OF, HEART, MYOCARDIUM AND ENDOCARDIUM (*Effects of Cardiac Disease, Sudden Death*), MEDICINE, FORENSIC (*Certificate of Death*), MEDICINE, FORENSIC (*Signs of Death*), MEDICINE, FORENSIC (*Death from Lightning, Electric Currents, Starvation, Cold, Asphyxia*), POST-MORTEM METHODS, PREGNANCY, DIAGNOSIS (*Death of Fetus*), PREGNANCY, INTRA-UTERINE DISEASES (*Death of Fetus*), PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Death*), PUERPERIUM, PATHOLOGY (*Sudden Death, Causes*), TEMPERATURE (*Pre-Agonic, After Death*), TOXICOLOGY (*Post-Mortem Appearance of Poisons*), TYPHUS FEVER (*Death in*), VITAL STATISTICS (*Deaths*), etc, etc.

**Death-Rate.**—The ratio between deaths and population, stated (commonly) as so many deaths (e.g. 14) per annum per 1000 of the population. See VITAL STATISTICS (*Deaths*).

**Death-Rattle.**—The rattling sound caused by the breath passing through mucus in the throat of the dying, is not an invariably certain sign of impending death.

**Death, Signs of.**—The distinction between the quick and the dead is not always so absolute as might at first appear. Long after what is called the moment of death vital phenomena continue in some of the tissues of the body, and even if we set aside all cases of Indian fakirs and Colonel Townsends, there remains a sufficient residuum of cases of persons presumably dead afterwards returning to life to raise in the minds of some a morbid fear of being buried alive. The conditions which may thus most nearly resemble death are syncope, asphyxia, and trance.

The principal signs relied on as proving that death is real and not apparent are—

1. *Entire Cessation of Circulation and Respiration.*—The entire cessation of all respiratory movement for a period of five minutes is itself sufficient proof that death is real and not apparent, but the test of auscultation should be applied for a sufficient period to make sure that the action of the heart (*primum vivens et ultimum moriens*) has really finally ceased. The popular method of testing for the continuance of respiratory movements consists in holding a cold mirror or a fluff of cotton before the nose and mouth.

If respiratory movements are still going on, the mirror will be dimmed or the fluff of cotton by its movement will betray the passage of the air. To test the cessation of the circulation it has often been suggested that a small vein should be opened. A nenter test is that proposed by Magnan, who recommended that a ligature should be tied firmly round a limb. If circulation was going on even slowly the limb beyond the ligature would gradually become congested.

2. *The Cooling of the Body*—Under ordinary circumstances a dead body is quite cold in from twelve to twenty hours after death. The rate of cooling is, however, modified by many circumstances. Thus a fat body will cool less quickly than a thin one. A body naked or exposed to cold air will cool more quickly than a body wrapped in woollen coverings or surrounded by a warm atmosphere. In water a body will cool more rapidly than in air of the same temperature. It is said that when death occurs from hyperpyrexia the temperature may even continue to rise for a short time after death.

3. *Cadaveric Rigidity—Rigor Mortis*.—After death the muscular system typically passes through three stages. First, there is a stage of flaccidity in which the muscles retain then power of contracting on electric stimulation. Muscles which were contracted in the act of dying may pass into a condition of cadaveric spasm instead of relaxing at "the moment of death." The second stage is that of rigor mortis. The third is that of relaxation and incipient putrefaction. Rigor mortis usually sets in six or eight hours after death, and may last for three to six or seven days. Its usual duration is about twenty-four hours. It commences in the muscles of the neck and lower jaw, and passes on into the muscles of the upper limbs, chest, and lower limbs. It passes off in the same order. Rigor mortis is a phenomenon analogous to the coagulation of the blood. It is due to the coagulation from the muscle plasma of a protein termed myosin, and the process is accompanied by the liberation of carbonic acid, and a change from alkaline to acid in the reaction of the muscle substance. The period of onset and the duration of rigor mortis depend chiefly on the degree of exhaustion of the muscular tissue. When death has occurred from convulsions or by accident, for example, during prolonged and excessive muscular exertion, rigor comes on very rapidly and passes off correspondingly quickly.

4. *Hypostasis*.—The gravitation of the blood to the most dependent parts of the body results in the post-mortem staining which begins to appear in from eight to twelve hours after death. If the body has been lying on a hard table the parts in actual contact with the table, such as the buttocks and scapular regions, may be quite white, while the parts immediately surrounding are deeply stained. Hypostasis must not be

confounded with ecchymosis, in which condition the blood is actually extravasated into the subcutaneous tissue. The distinction in a doubtful case is easily made by making a small incision into the discoloured part.

5. *The Lustre of the eye* is very quickly lost after death, and the tension of the globe falls so that the cornea feels flaccid. These changes occasionally take place even before death, for example, when death is due to malignant cholera.

6. *The Skin*.—After death the skin loses its normal elasticity. It undergoes striking alterations in colour. The first change is a general pallor of the whole surface. A little colour may, however, be left in dilated venules. Ecchymoses, of course, retain their colour. Later, the discoloration due to hypostasis appears, and this is followed by the colour changes associated with putrefaction.

7. *Putrefaction*.—As rigor mortis passes off the putrefactive changes which end in the complete dissolution of the body make then appearance. The first signs of putrefaction are that the limbs become supple, a faint odour becomes noticeable, and a greenish discoloration appears on the abdomen, and later on the chest, face, arms, and legs. The progress of putrefaction may be limited to a greater or less degree under suitable circumstances by the occurrence of mummification or the formation of adipocere. It is said that putrefaction may be greatly delayed in the bodies of persons who have been arsenic eaters.

**Death Struggle.**—The *agonia* or the pre-mortem convulsive throes or pangs. See AGONY, POST-MORTEM METHODS (*Agonal Intussusception*).

**Debility.**—Weakness in general, or weakness of mind (*debilitas animi*) or memory (*debilitas memorie*). See NEURASTHENIA, HEADACHE (*Cause*), etc.

**Decapitation.** See LABOUR, OPERATIONS (*Embryotomy, Trunk Presentations*).

**Decapsulation, Renal.**—The splitting or separation of the capsule of the kidney as recommended and carried out by Edebohlis (1899) in cases of Bright's disease and (afterwards) in purpural clampsia, its value has not yet been established. It is sometimes associated with incision of the renal substance (nephrotomy).

**Dechery Cautery.** See CAUTERY (*Thermo-Cautery*).

**Dechlorination or Dechloridation.** See also URINE, PATHOLOGICAL CHANGES; DROPSY.—Dechlorination, or restriction of the amount of salt consumed to the minimum contained in the food elements, has recently been introduced into therapeutics in the treatment

of diseases attended by dropsy, and, in particular, Bright's disease. Thanks to the labours of Widal and others, the method has been elaborated on a rational basis, and incidentally much light has been thrown on the salt interchange of the body as well as on the nature of oedema. Contrary to popular belief, there is no evidence that the almost universal practice of adding salt to food is necessary to health, without labouring this point, it may be said that among the less civilised races there are many exceptions to the rule, and that the alleged instances of ill-health following privation from salt break down on careful scrutiny. The actual requirement of salt, as estimated by the amount excreted in fasting conditions, is about 6 gram daily, and this loss is more than replaced by the amount—1.5 to 2 grams—naturally present in an average mixed diet. Experiments show that in health the body is in a state of chloride equilibrium, the amount eliminated daily corresponding to what is ingested. All the tissues of the body are bathed in saline fluid, and it is imperatively necessary that the osmotic pressure, or concentration, of this should remain constant. The negative action of "physiological" salt solution on tissue cells as compared with the deleterious effects of stronger and weaker solutions is an illustration of this. Variations in the quantity of salt in the body must therefore be compensated for, and this is done, partly by the retention of enough water to keep it in solution of normal strength, and partly by the eliminatory action of the kidneys. If a healthy man be placed on a diet free from added salt he continues for several days to eliminate a quantity exceeding that ingested, losing weight the while, at the end of that time chloride equilibrium—the output equals intake—is restored, and the weight remains stationary. The addition of a fixed ration of salt causes the weight to rise again, there is a corresponding retention of chlorides as measured by the output before equilibrium is re-established. From such experiments it has been shown that about 12 grams of sodium chloride and 1.5 to 2 kilos of water represent the floating quantity lost by a normal subject in the process of dechlorination. Owing to the ability of the kidneys to excrete chlorides freely, large doses are tolerated in health, yet the power of even the normal organ has a limit, and the habitual consumption of excessive quantities of salt beyond the renal capacity has been known to lead to retention of salt, giving rise to oedema from the excess of water required to keep it in isotonic solution.

In many forms of nephritis the kidneys lose their permeability to sodium chloride, the salt is retained in the tissues, and dropsy ensues. This has been abundantly verified by experiment. Patients with Bright's disease, swollen with dropsy, when put on a diet containing no added salt often lose weight steadily, the

oedema also vanishing. On adding salt the weight rises, and the dropsy reappears. It is found in such a case that on each addition or subtraction of salt with its corresponding rise and fall in the weight curve there is a definite level at which visible oedema makes its appearance. Widal calls the interval during which the weight is rising towards this point the *pre-oedematous period*, it represents the accumulation of fluid in the viscera. We need not discuss the relation of salt retention to the syndrome of uræmia, the evidence that it is responsible for the nervous symptoms is less tangible than its causal connection with oedema. It must, however, be said that in removing oedema by dechlorination we have more than a symptomatic treatment. The kidneys, like the other viscera, suffer from the effects of interstitial oedema, and there is reason to believe that in curing this renal oedema we break a link in the chain of a vicious circle, in which the inadequacy of the kidney produces a renal lesion which still further impairs the efficiency of the organ. Whatever be the precise explanation, it is certain that a dropsical patient, whose kidneys are impervious to very small quantities of chloride, may, after dechlorination, regain the power of eliminating it in considerable amount, and with this there may be marked diminution in the albumen passed. Dechlorination is occasionally attended by nervous symptoms resembling those of uræmia, these, however, are rare and transitory.

The practice of dechlorination is simple, and, contrary to what might be anticipated, patients seldom rebel against being deprived of salt. Milk contains 1.6 grams of salt per litre, which is equivalent to about 5.6 grams in an average daily ration. Although this considerably exceeds the proportion naturally present in a mixed diet, it is much inferior to the quantity habitually consumed as added salt (15 to 20 grams), and probably the efficacy of milk in the treatment of nephritis is as much due to its comparative poverty in chlorides as to any special attribute of its protein content. In strict dechlorination, however, recourse must be had to a mixed diet, which may be selected from among the following, cooked without the addition of salt.—Fresh meat of all kinds, fresh-water fish, eggs, fresh butter, cream, potatoes, and farmacæ generally, sweets, vegetables, and fruit. The only article which requires special fabrication is bread, as ordinarily baked it contains from 8 to 1 per cent of salt, thus, therefore, must be omitted. Soups, sea-water fish, all forms of salted or preserved foods, cheese, and milk are proscribed. In practice Widal allows from 100 to 200 grams of meat daily, with farmacæ, etc., as the appetite demands.

Patients subjected to dechlorination should be kept in bed until the weight ceases falling and remains stationary. Should dehydration

be slow, it may be aided by diuretics, particularly theobromine. To gauge the amount of fluid (and chlorides) lost by the body careful daily weighing is essential, and replace chlorides estimations.

**Dechlorination** has been successfully employed in dropsical affections of all kinds, such as cardiac and hepatic disease, oedema of the legs from exertion, phlegmasia, etc. The rationale of its action is the same in all such cases. It has also been employed in epilepsy, but on quite other grounds, the hypothesis being that by rendering the tissues poor in chlorides the bromide introduced is enabled to act more effectively.

**Decidua.** See ABORTION (*Causes, Maternal, Decidual Disease*), CAUDA, ECTOPIC GESTATION, FETUS AND OVUM, DEVELOPMENT OF (*Decidua*), MOLE, MOLAR PREGNANCY (*Mola Carnosa*), PREGNANCY, OVUM AND DECIDUA (*Affections of the Decidua*), PREGNANCY, DISEASES OF PLACENTA AND CORD (*Diseases of the Membranes, Decidual Endometritis*).

**Deciduoma Malignum.** See ABORTION (*Incomplete*), CHORION, CHORION-EPITHELIOMA, FALLOPIAN TUBES (*Tumours, Deciduoma*), LABOUR, POST-PARTUM HÆMORRHAGE (*Secondary*), PUERPERIUM, PATHOLOGY (*Sarcoma - Decidua - Cellular in Deciduoma Malignum*), SANCYOMA, TUMOURS (*Epithelial Group*).

**Declaration, Dying.** See MEDICINE, FORENSIC (*Dying Declarations*).

**Declination.**—Deviation, also the remission of a disease or of the paroxysm of a disease.

**Decline of the Birth-Rate.**—A phenomenon of the close of the nineteenth and the beginning of the twentieth centuries, affecting all civilised countries, due doubtless to several causes, but especially to the postponement of marriage till late in life (opportunity) and to the use of checks to prevent conception and pregnancy. In Edinburgh in 1871 the birth-rate was 34.89 per 1000, in 1881 it was 32.23, in 1891 it was 28.25, in 1901 it was 24.99, and in 1905 it was 22.99.

**Decocta.**—Solutions of vegetable drugs made by boiling in distilled water for 5 to 10 minutes, and then straining, there are three official decocta (*Decoctum Aloes Compositum, Decoctum Granati Cortex, and Decoctum Hamamelidis*), and the dose of each is  $\frac{1}{2}$  to 2 fl oz.

**Decolator.** See LABOUR, OPERATIONS (*Embryotomy, Decapitation*).—An instrument shaped like a hook (sharp or serrated) or like a key, used for decapitating the foetus in impacted

shoulder cases in labour, a decapitator (*e.g.* Braun's).

**Decomposition.** See MEDICINE, FORENSIC (*Signs of Death, Putrefaction*).

**Decubitus.**—The attitude adopted by a patient, particularly in bed, posture in bed, also means a bedsores (as in myelitis).

**Decussation.**—A crossing of structures (especially of nerve fibres) in the form of a X, *e.g.* the decussation of the pyramids of the medulla oblongata, of the fillet, or of the pons, chiasm.

**Defaecation.** See CONSTIPATION (*Physiological Considerations*), DIGESTION AND METABOLISM (*Defecation*), PHYSIOLOGY, FOOD AND DIGESTION (*Defecation*).

**Defectio Animi.**—Mental deficiency, deliquium animi.

**Defectus.**—Defect or absence of development of any part, *e.g.* defectus vaginae or atresia vaginae.

**Defervescence.**—The stage of decline of a fever, which is either short ("by crisis") or protracted ("by lysis"), the temperature and pulse-rate fall, the skin acts freely, and there may be a profuse diuresis or a diarrhoea.

**Defibrination.**—The process by which the fibrin is removed from the blood.

**Defloration.**—The tearing of the hymen and consequent destruction of the signs of virginity in a woman. See FORENSIC MEDICINE (*Rape*).

## Deformities.

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#### ARTHRODESIS

**Definition**—Arthrodesis is an operation designed to fix a joint in a case of entire paralysis of the muscles in the neighbourhood of that joint. Its object is ankylosis either fibrous or osseous, and it is obtained by complete removal of the articular cartilages.

**Indications for Operation**—1 When the paralytic condition of the limb is very severe. 2 In poor patients who are unable to provide themselves with apparatus. 3 When an apparatus is hardly tolerated and causes pressure-sores. 4 Not in spastic cases nor in paralytic cases after acute febrile disorders, as paralysis especially in zymotic diseases shows a strong tendency to recovery. 5 When two joints of a limb are hopelessly flail-like it is advisable to fix one or both of them.

**Methods**—When the operation is done at the knee or at the ankle, the two joints most favourable, the essential part of the proceeding is the total removal of the articular cartilage in such a way that the bone surfaces are left in contact.

**At the Knee**—The usual method is as follows, and is carried out with all aseptic precautions. An incision is made transversely across the joint from one condyle of the femur to the other, and passing across the middle of the patella. The patella is sawn through, and is then turned upwards and downwards. The articular cartilage is removed from the back of the patella. The joint is now fully exposed and treated as follows.—All the articular cartilage is carefully cut away with a gouge, scissors, and sharp spoon or knife. From the anterior surfaces of the tibia and of the femur it is also advisable to take a slice of bone off vertically, so as to fix the posterior surface of the patella denuded of its cartilage to them. The bones are now wired together in such a way that the wire passes through the lower fragment of the patella, then through the tibia, the femur, and through the upper fragment of the patella. The limb is now placed in the straight position, and should be kept immobile by plaster of Paris for at least two months, and after that a posterior splint is sufficient.

**At the Ankle**—Considerable difference of opinion exists as to the most convenient incision for the ankle, whether it should be anterior, posterior, or external. On the whole, the anterior method gives the most room. Against the anterior incision there are urged the section of the artery with deprivation of the foot of a part of its blood-supply, and the difficulty of getting the tendons to unite satisfactorily. But in practice these objections are not serious. The incision is made across the front of the joint from one malleolus to the other, dividing all the soft structures. The joint is thus freely opened, and the articular cartilages completely removed. It is not necessary to dissect away every piece of the synovial membrane, since in those cases in which it has been left its presence has not interfered with the formation of bone. But no loose pieces of cartilage are to be left in the wound, and none attached to the surface of the bone. It is often advisable to freely "stipple" the bony surfaces. The tendons are then united and the wound closed. The foot is placed in plaster of Paris at a right angle for six weeks. By this time firm fibrous union has occurred. To ensure osseous union, a difficult proceeding, the bones have been united by catgut, kangaroo-tendon, or ivory pegs. But absolute osseous ankylosis is not necessary, and is rather to be deprecated at the ankle. Close fibrous union allowing about 10 degrees of movement gives the best possible result. In cases of paralytic talipes calcaneus the best incision is a curved one beginning behind the peronei tendons, running down to the point of the heel and then upwards to the posterior edge of the flexor longus pollicis. The flap so made is lifted up with the tendo Achillis in it, and the opportunity is thus given of shortening the tendon. The joint is then freely opened and the cartilage removed.

**Results**—The result of the operation is to render a flail-like limb comparatively secure, and to lessen the number and weight of apparatus required. No fear need be entertained of difficulty in obtaining primary union in these paralytic limbs.

#### CONGENITAL DISLOCATIONS

##### *Congenital Dislocation of the Hip—Etiology*

—It is more common in the female than in the male sex, and is also hereditary. The number of unilateral and bilateral cases is about equal. One important factor in its production is a breech presentation, and if this be combined with a faulty acetabulum then the head of the femur is easily displaced at birth.

**Pathology**—*The Acetabulum*—In some cases it is entirely wanting, while its site is occupied by a convex mass of bone. In other cases the cotyloid cavity is present, but is defective. It may be found small and shallow, and about one inch in diameter. In shape it is sometimes

circular, but more often triangular, owing to non-development of the ischial part of the Y-shaped cartilage. Its shallowness is also due to defective development of the cotyloid rim, especially at its postero-superior border. The upper extremity of the femur is normal at birth, but as age advances the head undergoes flattening in front and above, and the neck becomes considerably shortened and less obtuse, and further is anteverted or retroverted. The *round ligament* is occasionally thick and solid, but more often elongated and attenuated or even merged into the joint-capsule at the anterior part. In 83 per cent of the cases it is absent. The *capsular ligament* is always elongated, and sometimes is remarkably thickened, while at other times it is seen to be dilated and thinned. Sometimes it is of a curious hour-glass shape, and is obliterated in its middle. As to the *muscles*, the ilio-psoas undergoes such a change in its direction that it finally comes to lie internal and even posteriorly to the head of the bone. The displacement of the caput femoris is usually above and posterior, but as the subject is more carefully studied the number of anterior dislocations is found to be large. The *symptoms* are as follows.—A typical rolling gait, lordosis, prominence of the abdomen, shortness of the lower limbs in proportion to the general bodily development, and feeble development of the muscles of the lower extremity. When the patient stands the great trochanters are unusually prominent, and displaced forwards and upwards or forwards and backwards. The heels also are rotated inwards and the toes turned outwards. When the patient is placed recumbent, the lordosis disappears and the tops of the trochanters are not so near the crests of the ilia as when he is standing. The upper borders of the trochanters are in all cases above Nélaton's line. The limb can often be made to "telescope" up and down. Signs which are absent are: pain and limitation of movement, especially in young children.

*Prognosis*.—As to the possibility of cure, the earlier treatment is commenced the more likely it is to be successful, and if it be begun before the child is four years of age it probably will be so entirely, but after that the prospect is doubtful no matter what form is adopted.

*Diagnosis*.—It is essential to make sure that the deformity is truly congenital, i.e. has arisen from causes *in utero* and not to unskilful and violent delivery. Paralytic dislocation should also be excluded. Coxa vara is diagnosed from congenital dislocation by the facts that in the former the limb cannot be pulled down by traction, and there is no flexion of the thigh and no muscular wasting. Pseudo-hypertrophic paralysis has often been confounded on superficial observation with congenital dislocation on account of the lordosis and waddling, but a little care suffices to distinguish between them.

Tuberculous coxitis has been confused with it, but the pain, limitation of movement, with loss of complete flexion, are quite sufficient for differential purposes.

*Treatment*.—The difficulties which have to be overcome are the ill-developed acetabulum, the alteration in the shape of the head, of the femur, and in the direction of the neck, the stretched condition of the capsule, and the contraction of the adductor muscles.

*Prolonged rest with extension* has been advocated by Buckminster Brown and William Adams, who have published several cases, but it must be confessed that grave doubts have arisen as to the permanency of the alleged cures. The time involved in treatment is at least four years, two of complete recumbency and two of walking on crutches. The disadvantages of this method are evident. Schede has modified this by placing the patient in the recumbent position, applying extension to the abducted limb and moderate lateral pressure on the trochanter. He does this for three months, and then allows the patient to get about on the sound leg with an outside steel support for the affected limb. In this support there is an abduction screw, by turning which pressure may be made on the trochanter. This treatment is suitable only for children under three years of age. Lannelongue, in addition to the rest and recumbency method, has injected two or three minims of a 10 per cent solution of chloride of zinc with the object of causing firm fibrous growth around the head of the femur.

*Treatment by reduction*, as initiated by Pagni and modified by Lorenz. The method is as follows. The patient is anaesthetised fully, and placed on a low couch so that the surgeon can stand well over him. The limb is then extended either manually or by means of pulleys until the top of the trochanter is at or just below the Nélaton line. If the muscles refuse to allow the trochanter to be drawn down they are divided subcutaneously. The limb is now fully abducted, and by a little judicious rotation the head of the femur may be felt to slip over the posterior upper margin of the acetabulum, and it sometimes moves in with a distinct click. The difficulty now is to keep it fixed. This is best effected by placing the thigh at about 75 degrees of abduction and using plaster of Paris. In about three weeks the patient is allowed to walk with the leg in this position. Two or three inches of cork-sole are added to the shoe on the sound side, and the assistance of a crutch or crutches is useful. The object of keeping the limb in this abducted position is to wedge it firmly into or upon the acetabulum. The plaster of Paris is renewed at the end of three months and reapplied for a second period.

*Treatment by open operation*. The original method of Hoffa has now been discarded, and Lorenz has modified it. But it is to be noted



that when surgeons of great operative skill and experience have endeavoured to follow in Lorenz' footsteps they have met with nothing like the success he claims. Arbuthnot Lane states that he has had good results by fixing the head close behind the anterior inferior spine.

*Other Joints*—*Congenital dislocations* of almost every other joint of the body have been met with, but they are merely surgical curiosities of no practical interest.

#### CEREBRAL AND SPINAL PARALYSIS, DEFORMITIES ARISING FROM

The forms of cerebral and spinal paralysis which give rise to deformities are acute anterior poliomyelitis, of the cerebral and spinal forms, infantile hemiplegia and spastic paralysis of childhood, locomotor ataxy and Friedrich's disease, syringomyelia, compression paraplegia arising from causes of the spine.

*Deformities arising from Acute Anterior Poliomyelitis of Cerebral and Spinal Origin*—The deformities met with in the trunk are scoliosis, kyphosis, and lordosis of paralytic origin. Of these the most interesting is scoliosis, and this is of an extremely intractable form, but it must be treated by massage, electricity, and supporting apparatus. The deformities of the arm arising from poliomyelitis are several. The deltoid is usually wasted, and there is often a paralytic subluxation of the shoulder-joint. In the "fore-arm" type of paralysis of Renak the extensor muscles are paralysed, while the supinator longus escapes. Wrist-drop then results. Occasionally the adductor muscles of the thumb are affected.

When the *lower extremities* are completely paralysed they are small, cold, and bluish, perfectly limp, and swing like a flail in all directions. The joints are so lax that the segments of the limbs may be made to assume almost any position. It is important to ascertain in these severe cases of paralysis of the lower limbs if the psoas have or have not escaped, for so long as the psoas retains power the patient can be made to walk with instruments after tenotomy and when any contraction has been rectified. Paralytic subluxations are met with either in the hip, knee, or ankle.

*Treatment of Paralytic Deformities*—In the first place every effort should be made from the time of onset of the paralysis to retain the limbs in as nearly normal a position as possible. That is to say, it is important to anticipate the deformities. Electrical stimulation of the muscles, friction, and massage should be assiduously persevered with. The active treatment of those deformities must be of two kinds—mechanical and operative. The objects of mechanical treatment should be to support and protect the paralysed limb in such a way that the muscles shall work to the best advantage and that the joints are supported and controlled, also to overcome by means of suitable apparatus deformities

which have already occurred, and to prevent their recurrence. The operative treatment consists in tenotomy and fasciotomy, osteotomy, arthrodesis, and tendon-transplantation. Tenotomy is useful in relieving the various forms of talipes of the foot, the contraction of the knees, and the flexion of the thighs. Osteotomy is not often called for. The indications for arthrodesis have been given in the article of that heading (p. 279), and the indications and methods for tendon-transplantation are treated under that title (p. 282).

*Infantile Hemiplegia and Spastic Paralysis*—Deformities of a most persistent and intractable nature often ensue from these causes, and the arm is more affected than the leg. The position assumed by the arm is quite typical. In this limb flexion predominates. The shoulder is sometimes raised and sometimes lowered. The upper arm is generally kept parallel with the trunk. The forearm is flexed at a right angle, and is generally in apposition with the lower part of the chest or the upper part of the abdomen. It is almost invariably pronated, very rarely supinated. The wrist is strongly flexed, and the hand is adducted. The fingers are firmly contracted. The leg is in a condition of extension, and the foot assumes a talipede form, either of equinus or equino-varus. The mode of walking is thus: the heel is raised and the foot is lifted from the ground with difficulty, the toes scrape along the floor, and later the patient becomes scissor-legged in progression. The treatment is as follows.—Every effort must be made, if the case be seen early, to prevent the onset of deformity by massage, manipulation, passive movements of the joints. The patient should also be taught to use the affected limbs as much as possible, and much improvement may be expected in slighter cases. With reference to the use of retention apparatus, the writer's experience is that directly it is removed the spasm returns. With regard to operative measures in the upper extremity, the writer has lately devised and successfully carried out an entirely new form of operation. It is as follows.—The tendon of the pronator radii teres is exposed and separated from its insertion into the radius. A gap is made in the interosseous membrane, and the pronator radii teres is carried round *posteriorly* to the radius, and is reinserted at its normal site. It therefore becomes a supinator instead of a pronator. The flexor carpi radialis and other tendons at the wrist are divided so that the wrist-drop is overcome, also the contraction of the fingers. In the lower extremities operative measures are fully indicated. For spastic talipes the tendo Achillis should be severed, for contracted knees the hamstrings, and in the thighs the tensor vagina femoris, the sartorius, and adductors should be divided. It is astonishing the amount of good that results from this proceeding.

*Locomotor Ataxy*—This causes spinal de-

formities very rarely. Sometimes the deformity is angular, or sometimes scoliosis is present. In Friedreich's disease or congenital locomotor ataxia, scoliosis develops late in the disease, and is generally seen to be with the curve to the right in the dorsal region. In this disease talipes arcuatus is also met with, and is spoken of later. Progressive muscular atrophy gives rise to a peculiar form of talipes varius, due to peroneal paralysis. In syringomyelia various distortions of the spine may be met with, and many forms of contractions of the limbs.

*Compression paraplegia* is a frequent complication of spinal caries. It is rarely due to pressure on the spinal cord by bone, but more frequently it is caused by a tuberculous pachymeningitis, the thickened membranes constricting the cord. The paralysis is usually bilateral. In rare cases it is unilateral. It affects the legs generally, although the arms may suffer later, or both may be paralysed simultaneously. It occurs in about 1 in 12 to 15 spinal cases. The symptoms of compression paraplegia are as follows—Its onset is in some cases sudden, but more often it is gradual. As to the motor functions, the patient complains of getting tired easily, and soon the legs begin to drag and the toes to catch in walking. Dull aching pain is common in the early stages in the body and limbs. In the body the most usual form is girdle pain or pain in the pit of the stomach. These pains are due to irritation of the nerve roots. Oftentimes there is no anesthesia, or it occurs occasionally without motor symptoms. The reflexes are exaggerated both superficially and deeply. The sphincters are often involved, and incontinence of urine and feces occurs in severe cases. The affected muscles waste, and the reaction of degeneration is more or less marked. The limbs are often cold, but sometimes perspire persistently. The diagnosis is not as a rule difficult when the back is examined. Sometimes it happens that compression paraplegia sets in before deformity has appeared. The prognosis of these cases is that a great number of them show a strong tendency to complete recovery without operation. Mere recumbency with extension is often sufficient to induce an immediate change for the better. Recurrent attacks are dangerous, but not so *per se*. Cystitis and bronchitis are grave complications. With regard to treatment, prolonged recumbency with extension of the spine in the horizontal position often effects a surprising improvement, and should be persevered with. But if, after eighteen months to two years of this treatment, the symptoms do not improve but rather get worse, then laminectomy should be resorted to. Too great results must not be expected from laminectomy, for the operation is as often a failure as it is a success. Forcible rectification of the deformity has relieved some troublesome cases of paralysis.

#### RICKETS—DEFORMITIES OF

The skull is often enlarged and thin, and with it there is associated hydrocephalus. The fontanelles remain open. The forehead is square and the upper wall of the orbit is oblique. There is also a peculiar development of the lower jaw, its alveolus is somewhat inverted, so that the teeth point inwards. With regard to the spine, the rickety deformities are kyphosis and, later, scoliosis. The chest, too, is the subject of deformity, pigeon-breast, exaggeration of the curves, and subluxation of the inner end of the clavicle. Rickety deformity of the arm is sometimes seen in severe cases, and both bones of the forearm may be bent inward or outward, or one bone alone. The rickety pelvis is important to obstetricians, and the conjugate diameter is decreased owing to the prominence of the sacro-vertebral angle. In consequence of the inward thrust of the head of the femur into the acetabulum on each side, the lateral aspect of the pelvis is flattened, and the pubic arch diminished. The rickety deformities of the long bones of the lower extremity are fully described in the sections dealing with deformities of the knee and foot.

#### TENDON-TRANSPLANTATION

By this is meant the reinforcement of a paralysed muscle by attaching to it the tendon of a healthy muscle. It is also known by other names, as muscle-grafting, function transference of tendon, but the word tendon-transplantation sufficiently indicates the scope of the procedure. Before deciding to perform tendon-grafting there are certain points to be observed, namely—1. The muscles should be carefully tested electrically, and the relative strength of each of them determined, 2, all conditions due to contraction of the plantar fascia, such as pes cavus, should be previously removed by operation, 3, the operation is rarely called for when one muscle only is partially paralysed, 4, cases in which all the muscles are paralysed are suitable for arthrodesis only, 5, in selecting a healthy muscle for reinforcing a paralysed muscle it is advisable that the one selected should belong to the same group, if possible, as the paralysed one, and the muscle selected should also be, as far as may be, in the same line as the paralysed muscle, 6, the reinforcing tendon should be carried as directly as possible to the paralysed muscle and not bent round at an angle. Tendon-transplantation is particularly applicable to talipes calcaneus, as the peronei muscles and the posterior tibial deep muscles, i.e. the tibialis posterior and the flexor longus digitorum and the flexor longus pollicis, are very suitable for grafting into the tendo Achillis. Cases of simple equinus do not as a rule call for muscle-grafting, because division of the tendo Achillis is sufficient. Cases of paralytic valgus due to paralysis of the tibialis

anticus and posticus may be treated by transplantation of the extensor proprius pollicis into the tibialis anticus and a piece of the flexor longus digitorum tendon into the tibialis posticus. On similar lines talipes varus may be treated. As to the actual method of procedure, the operation requires that the parts should be absolutely aseptic. The tendons to be operated upon are exposed, and their sheaths freely opened in such a way that the tendon of the reinforcing muscle can be applied easily to the paralysed muscle. Taking, e. g., the fixing of the peroneus longus to the tendo Achillis, the sheaths of the peronei and of the tendo Achillis are freely opened up and a considerable portion of the peroneus longus tendon is detached. It is then divided across just above the malleoli and passed to the under surface of the tendo Achillis nearly opposite the ankle-joint, the tendo Achillis having been previously split longitudinally at this spot. The proximal end of the peroneus longus tendon is drawn through it and split somewhat, and the ends attached to the posterior surface of the tendo Achillis by being sewn down with silk or some other material. The proximal part of the tendon of the reinforcing muscle should be pulled quite tightly, and inserted at such a spot in the tendon of the paralysed muscle that the former, when it is fixed, is at its utmost possible tension. The wound is then allowed to heal and the strength of the reinforcing muscles is subsequently increased by massage and galvanism.

#### HAND AND FINGERS

**CONGENITAL DEFORMITIES**—These comprise *club-hand*, *congenital contraction of the fingers*, *supernumerary fingers* or *polydactylism*, *suppression of the fingers*, *web-fingers* or *syndactylism*, *hypertrophy of the fingers*, *congenital lateral deviation of the fingers*.

**Club-Hand**—This is a very rare deformity, and there are comparatively few cases recorded in literature. In most cases the child is both premature and still-born. The forms of club-hand are as follows, and it should be remembered that the hand may deviate either to the outer or inner border of the forearm, or be in a position of flexion or extension. Hence we have radial and ulnar club-hand and palmar and dorsal, so that there are the radio-palmar, radio-dorsal, ulnar-palmar, and ulnar-dorsal. In the palmar cases the hand forms with the forearm a more or less acute angle open anteriorly. The lower end of the radius is prominent posteriorly, and the carpus articulates with the anterior surface of the radius. There is generally some degree of mobility of the hand on the forearm. It is small and wasted owing to the shrinkage of muscles and the absence of some parts of the bones.

**Treatment**—The means which are adopted to alleviate the deformity are passive movements,

massage, the use of retentive apparatus, tenotomy, and operations on the bones. In any case the result is not very satisfactory.

**Congenital Contraction of Fingers**—This deformity is quite distinct from contraction of the palmar fascia known as Dupuytren's contraction, and should not be confused with it. The affection is generally limited to the fifth finger, but at times the ring finger and even all the fingers are contracted. It is often associated with congenital hammer-toe, and in that event the second toe is the one usually affected. Some contraction of the finger is frequently met with, but it is only when it gives rise to pain and annoyance that any notice should be taken of it. In congenital contraction the first phalanx is usually hyper-extended, and the second and third are flexed, in contradistinction to Dupuytren's contraction, in which the first and second are flexed and the third is generally extended. If the deformity is left unretained, contraction of the skin and fascia and of the lateral ligaments of the inter-phalangeal articulations takes place. But the contracted fascia never extends up into the palm as in Dupuytren's contraction.

**Treatment**—In the milder degrees it is sufficient to straighten the affected fingers by frequent passive movements, and to maintain the correction by the use of a small malleable iron splint adapted to the dorsal surface of the fingers. This often suffices to remove the deformity. Should it fail to do so, the contracted fascial bands may be divided and the finger put up in a suitable splint. Unfortunately these cases have a strong tendency to relapse, and it is well to warn the patient that after a finger has been straightened a long course of mechanical treatment is necessary to maintain the improvement. For this purpose the apparatus similar to that used after operation for Dupuytren's contraction must be worn day and night for three months.

**Supernumerary Fingers—Polydactylism**—There are five varieties of polydactylism. (1) An additional finger is more or less developed, generally on the ulnar border of the hand, being attached to it by a narrow pedicle. (2) An additional thumb is more or less developed. (3) A supernumerary digit more or less perfect is closely united throughout its whole length to another digit. (4) A completely developed extra digit is formed, and possesses its own separate functions and tendons. (5) The bifurcated hand which has eight fingers and no thumbs.

**Treatment**—In the first variety the additional digit should be removed early in life. In the second variety removal is indicated too. As to the third variety the propriety of operation is doubtful. In the fourth variety it is advisable not to interfere, and the fifth admits of no treatment.

**Suppression of the Fingers**—These are of interest rather to the teratologist than to the surgeon. If the hand is very unsightly or useless, amputation of it is the best resource, and a good artificial hand is to be preferred.

**Webbed Fingers; or Syndactylism**—There are three varieties (1) Two fingers, generally those on the inner side of the hand, are united by skin and fibrous tissue (2) The union is by muscular as well as fibrous tissue and skin (3) The bones are fused throughout their whole length, or more often at the second and third phalanges only

**Treatment**—In the second variety little can be done, and the case is often best left alone. In the first and third varieties the chief difficulty after operation is to prevent some reformation of the web, especially towards the base of the new cleft. This difficulty can be overcome either by the formation of a permanent opening at the bottom of the web by transfixing it with a silver pin or by Didot's operation, or by those operations known by the names of Zeller and Norton. Choice of operation—(1) If the web is small and thin, or if the union between the fingers is very close, the formation of a permanent opening at the base of the web is to be preferred. The web may be divided subsequently, and its edges trimmed and sutured. (2) If the web is extensive, complete, and of good width, Didot's operation is the best. (3) If the web is incomplete and reaches but halfway, Zeller's or Norton's operation is indicated.

**Hypertrophy of the Fingers**—This condition is sometimes seen at birth in a minor degree, and becomes exaggerated later. The hypertrophy may consist of general overgrowth of all the tissues of the finger, lymphatic enlargement of the subcutaneous tissue, or a nevusoid condition of all the soft structures.

**Treatment**—Compression of the fingers and ligature of the arteries have been both tried, but without success. When the finger becomes a source of annoyance it should be removed.

**Congenital and Lateral Deviation of the Fingers**—This is very rare indeed, but is comparable to a condition seen in the toes. It may be treated either by a suitable apparatus, or by division of the lateral ligaments, or by amputation.

**ACQUIRED DEFORMITIES.**—Under this heading are comprised *Dupuytren's contraction*, *spring finger*, and *mallet finger*.

**Dupuytren's Contraction**—**Definition**—A permanent flexion of one or more fingers arising from contraction of the palmar fascia and its digital prolongations.

**Occurrence and Etiology**—It is many times more frequent in men than in women. As a rule the ring and little fingers are affected, but it may spread to other fingers. In some

cases it is distinctly hereditary. The age at which it comes on is generally in middle or in late life—that is, when fibroid changes supervene. Occupations seem to have some share in its production, especially those that involve repeated traumatism to the palm of the hand, as in gardeners, drivers, engravers, etc. In some instances it would appear as if the contraction began after a definite injury to the palm—a slight wound, a sudden bruise, or an excessive strain of the structures. Certain general conditions seem to predispose to it, as, for instance, gout and rheumatism, or the contraction appears to be much more common in gouty and rheumatic people, and to follow sometimes an attack of gout. Other people have noticed it to be associated with syphilis, and Mr William Anderson has hazarded the opinion that it is of bacterial origin.

**Its morbid anatomy** is, as follows.—The affection is primarily a contraction of the fascia, and secondly of the skin. The tendons have nothing to do with it. The palmar fascia is not a well-defined aponeurosis, but fades off gradually at its edges and gives off two sets of processes, the superficial to the skin and the deep to the lateral aspect of the fingers, passing to the sides of the first and second phalanges, and to the periosteum and to the tendon sheaths. The nature of the morbid change in the fascia is a fibroid hypertrophy. Sometimes this appears in the form of small fibromata, in others there is a general thickening of the bands, followed by contraction. Crystals of urate of soda have been found in the thickened fascia.

**Symptoms**—At first there is a feeling of tightness in the palm of the hand and in the ring or little finger, and the patient finds some difficulty in fully extending the fingers. Later there appears nodular indurations with adhesions of the skin. The latter is first seen usually in the transverse crease of the palm. The affected fingers then begin to retract, and the first phalanx flexes on the metacarpal bone.

**Treatment**—No measures except operative are of any avail. The operative measures are either multiple subcutaneous division of the contracted band as practised by Mr Adams, or open removal by a careful dissection of the palm of the hand. The method to be preferred is the multiple subcutaneous puncture. The operation may have to be repeated two or three times. After the operation a Dupuytren's splint is worn, and the fingers are gradually straightened. The result is as a rule very good, and relapses are not frequent. The writer has performed the open method several times in hospital patients, who cannot afford time to attend frequently, and he has been satisfied with the result.

**Spring Finger**, known also as *Jeil* or *Snap Finger*—The description of this affection is that if the patient closes all the fingers on the palm,

on opening them he finds that one remains shut, and it can only be extended by the other hand, and flies open like a knife-blade with a snap. Sometimes there is difficulty also in flexing the finger, which is accompanied by a small jerk. The affection is usually seen in the thumb, and is due to one of two causes, either a thickening of the tendon as it passes through its osseofibrous groove, or else narrowing of the groove. *Treatment*—If the thickening can be felt it should be cut down upon, and the thickened portion removed. In many cases, however, it is sufficient to blister frequently and the thickening disappears, or the finger may be fixed in a metal splint with pressure over the spot where the movement of the tendon is hindered.

*Mallet Finger*—This is also known as "drop finger," and is due to subcutaneous rupture of the extensor tendon where it is inserted into the last phalanx. Its usual cause is a fall forwards on the fully extended fingers, with the result that the fibres of the extensor tendon are torn away and the finger cannot then be fully extended. With regard to treatment, the finger may be placed in a malleable iron splint, with the last phalanx hyper-extended, or an incision should be made over the dorsum of the last phalanx, and the tendon reunited to the bone.

#### HALLUX VALGUS (BUNION)

It is largely due to the use of improper boots, not necessarily of tight ones, but of those which are pointed and often too short. In some cases it is due to osteo-arthritis and gout. The deformity is a displacement of the great toe outward, with prominence of the base of the proximal phalanx and of the head of the first metatarsal bone, mostly of the latter. Both these portions of bone are often enlarged and covered by a bursa or bursa. The bony swelling is especially noticeable in cases of osteo-arthritis. The anatomy of the affection is a partial subluxation of the first phalanx outwards from the head of the first metatarsal bone, leaving it exposed to pressure. The ligaments on the inner side of the joint are stretched, and occasionally perforated, those on the outer side are shortened. Of the tendons the extensor proprius pollicis is displaced outwards. The formation of the false bursa or bunion is secondary to the enlargement of the bone. The bunion is very liable to inflammation, and suppuration may cause cellulitis with occasional disorganisation of the joint. Sometimes the bunion has a corn on it. The symptoms are sufficiently plain, but the affection varies in degree, and there is no doubt that women suffer more frequently than men.

*Treatment*—Prophylactic. Pointed boots must be absolutely forbidden, and the inner edges of the boot should be quite straight. In slight cases curative treatment consists in wearing proper boots, with the application of cold

and soothing lotions to the inflamed and thickened skin, and the wearing of the digitated or divided socks. Some advise the use of a post between the first and second toes in the boot, but this does not answer well in practice. In other cases a bunion spring is used, but if the deformity is at all severe and the bursa large and painful, the only course is operation. It is not sufficient merely to chisel away the prominent portion of bone, but the operation *par excellence* for these cases is excision of the head of the metatarsal bone. With this operation the writer has ample reason to be satisfied.

*Hallux Varus or Pigeon-Toe* is the reverse of hallux valgus, but is not associated with pain in the same way. If it is excessive the treatment consists in manipulation and the use of a light splint to press the toe outwards.

*Hallux Rigidus* is a somewhat curious affection. The great toe cannot be extended beyond the straight line, and any attempt to do so produces severe pain. The tendon of the extensor proprius pollicis is always tense. With regard to the pathology of this affection opinions differ, but the writer has been able to ascertain that it is frequently associated with osteo-arthritis and with inflammation of the synovial fringe between the sesamoid bones. The mechanism of the affection is easy to understand. When an attempt is made to extend the toe, the head of the metatarsal bone and the base of the first phalanx press upon this inflamed spot so that the rigidity of the toe is really reflex. Local measures avail but little, and in cases which are at all marked it is always best to excise the head of the first metatarsal bone, and to remove a small portion of the prominent part of the base of the first phalanx.

#### HAMMER TOE

*Definition*—A deformity usually affecting the second toe, and consisting of dorsiflexion of the first phalanx, plantar flexion of the second, and extension of the third.

*Etiology*—In some cases it is congenital, and is associated with congenital contraction of the little finger. In other cases it is distinctly hereditary. But it is sometimes due to acquired causes. The chief of these is the use of short boots. The appearances presented are as follows. On the dorsal aspect of the first interphalangeal joint a painful corn is frequently present, beneath this is a bursa which from time to time inflames and suppurates. On the under surface the skin is contracted, and at the bottom of the groove the long flexor tendon can be felt. The first phalanx is in a state of extreme dorsiflexion, so that the head of the metatarsal bone is uncovered below to about half its extent. The anatomy of the part is as follows. In the first place there occurs contraction of the extensor tendon. This is followed by contraction of the flexor tendon, and with gradual shortening of

the lateral ligaments. The latter point is most important to recognise, as upon it depends successful treatment.

**Treatment**—In slight cases attention to the boots, together with manipulations and the use of the malleable iron splint at night, will remedy the trouble. But in many cases it is necessary to operate, and the writer would deny amputation of the toe, as it is quite possible to remedy the trouble without this rather severe procedure. If the toe be amputated, then bunion almost inevitably follows, because the first toe is readily displaced outwards. The operation consists in subcutaneous section of the flexor tendon and of the lateral ligaments from the under surface of the first interphalangeal joint, and often of the extensor tendon on the dorsum. After the operation the toe is fixed to a malleable iron splint until the small wound is healed, and it is then gradually extended. Good broad boots should be ordered for use afterwards.

#### TOES—DEFORMITIES OF

The other affections which are met with in the toes are *Syndactylism*, *Polydactylism*, *Suppression of the Toes*, and *Lateral Deviation of the Toes*. The latter is the result of bad boots, and may be remedied by manipulation, the wearing of a digitated sock, or by the use of a sole-plate with slots in it. In moderately severe cases the lateral ligaments may be divided, and in extreme cases amputation is necessary. Hypertrophy of the toes, generally of the first toe, is seen from time to time. The hypertrophied toe is frequently displaced inwards or outwards. It may attain an immense size. In most cases partial or complete amputation is required eventually. The treatment of syndactylism in the toes is the same as that of the fingers, although it may be better to amputate at once.

#### CLUB-FOOT—(CONGENITAL)

**Etiology**—The causation of congenital club-foot is still doubtful, although much light has been thrown upon it by the labours of R. W. Parker. The matter may be discussed under four headings: (*a*) arrested development of the bones of the leg, (*b*) causes arising from nerve lesions, (*c*) mechanical causes due to malposition of the foetus *in utero*, (*d*) abnormal development of the bones of the foot. (*a*) In describing bow-legs it is stated that certain forms of congenital bow-legs are accompanied by talipes equino-varus. Again, some cases of varus and equino-varus exhibit congenital absence of the tibia. But these congenital abnormalities are found in only a few of the cases of equino-varus, and cannot be the sole cause. (*b*) Nerve lesions. In some cases meningocele, encephalocele, hydrocephalus, and spina bifida coexist with congenital club-foot, but these are very few in proportion, so that the nerve theory fails to explain the majority of the cases. It has been held by some

that the deformity is due to partial asphyxia at birth, resulting in convulsions, but in the absence of definite examples, carefully collected and impartially examined, this opinion cannot be advocated. (*c*) Mechanical causes due to malposition or compression of foetus *in utero*. There is ample evidence to show that this is the probable explanation, and from the figures given by Parker in his work it is easy to understand that owing to intra-uterine packing the foot may during fetal life be retained in one position which becomes fixed. As a rule the position is such as to lead to some small degree of equino-varus, even in presumably normal infants, and they subsequently lose this tendency to eversion when they commence to walk. If the position is more decided, then an abnormal condition of the foot arises—either equino-varus in both feet, or equino-varus in one and equino-valgus in the other, or calcaneus in both. The most common modes of production are by accidental locking of the parts, by locking of the parts due to abnormal positions of the limbs, by exceptional positions of the limbs, independently of locking, and by congenital absence of certain bones. (The last supposed cause (*d*), abnormal development of the bones of the foot, cannot clearly be established, one can only say that the normal inversion of the lower limbs *in utero* persists some time after birth. The reason of the persistence is probably close intra-uterine packing, and this throws us back upon Parker's observations.)

**Forms of Congenital Club-Foot—Varieties**—The most common form is TALIPES EQUINO-VARUS. Some authors state that talipes varus is more common, but if a careful examination be made of a large number of cases it will be found that most of the so-called varus-cases have some shortening of the tendo Achillis and raising of the heel, so that really they are cases of equino-varus. And this point is proved by treatment, for until the tendo Achillis is divided, the foot cannot be fully rectified. The next most common form is calcaneo-valgus, while the pure calcaneus and pure equinus are rare, the last-named infinitely so. In the consideration of congenital club-foot, therefore, talipes equino-varus will be described fully.

**Appearances**—The nature of the deformity is as follows.—The heel is raised and the foot is extended, that is, it is in a position of plantar flexion. The sole of the foot and the toes are adducted and brought to the middle line instead of being directed to the front. The internal border of the foot is raised to a varying degree and bent upon itself, so that it is concave. With this the adduction of the great toe is often very marked. The external border of the foot is convex, and is in contact with the ground and forms the main point of support in progression. The deformity is situated in two places in the foot chiefly in the neighbourhood of the medio-

tarsal joint, and to a less degree at the tauto-metatarsal and phalangeal articulations and at the ankle. The recognition of the sites of deformity is essential to proper treatment, for the back part of the foot, that is behind the medio-tarsal joint, must serve as a fixed point for the correction of the front part, and fixation of the back part can only be obtained by leaving the tendo Achillis intact until the front part is restored. Congenital club-foot is more frequent in boys than in girls, and occurs about once in one thousand births. Double congenital valgus is more usual than single. Very frequently with club-foot of congenital origin, meningocele, spina bifida, partial or complete, amputation of the limbs, absence of the fingers, polydactylism, syndactylism, and absence of the bones of the leg or foot are found to coexist.

**Degrees**—The deformity varies according to age. **FIRST DEGREE**—The foot can be momentarily replaced by manual force. **SECOND DEGREE**

—The foot cannot be replaced manually. On attempting forcible reposition there remains some adduction or extension of the foot, and the sole cannot be planted squarely on the ground, and the great toe is much separated from the second. **THIRD DEGREE**—Seen in children and adults, the foot is in a rigid and resistant state with aggravation of the deformity and strong contraction of the soft parts. **FOURTH DEGREE**

—The deformity is inveterate and of old standing, and much malposition of the bones is present with the thickening of the soft tissues, and formation of false bursae on the outer side of the foot over the bony prominences. The foot may be so much distorted that the patient walks on the dorsum, and the sole of the foot looks upwards and backwards.

**MORBID ANATOMY**—Authors are agreed that in talipes equino-varus there exists extension of the astragalus upon the leg at the ankle-joint with twisting downwards and inwards of its head and neck, and inward subluxation of the scaphoid, elevation of the tuberosities of the os calcis, and rotation of the same bone around its vertical axis. The bones are modelled first of all in cartilage, and so long as they remain unossified they may still be moulded to a normal shape and no extensive operation is needed. Without discussing the minutiae of the morbid anatomy of club-foot it is necessary to remark that the angle of the neck of the astragalus is increased from thirty-eight to about fifty degrees. An astragalus taken from an adult case of club-foot presents several new features. Its body is no longer square but trapezoid, and even triangular, and the head protrudes from the front of the ankle-joint. The scaphoid is found to be much atrophied, and its tubercle has very nearly disappeared. On the anterior aspect of the internal malleolus is a new facet for articulation with the scaphoid. The ligaments on the dorsum and external border of the foot are elongated, and

those in the sole and on the internal border are contracted. The plantar fascia is also contracted. The tendons at fault are the tendo Achillis, tibiais anticus and posticus, extensor proprius pollicis, the flexor longus digitorum, and the flexor longus pollicis. Other somewhat remote lesions are met with associated with congenital club-foot. One interesting lesion is genu recurvatum with absence of the patella, another is scoliosis.

**The Obstacles to Reduction in Congenital Talipes Equino-Varus** arise (1) in the infant, from the altered direction of the neck of the astragalus, the contracted anterior fasciculus of the internal lateral ligament, the contracted astragalo-scaploid and calcaneo-scaploid ligaments. (2) in the adult, from the profound alteration in the shape of the bones, from the formation of new joints, from fixation of the ligaments and tendons in their abnormal attachments and course.

**Prognosis**—The points in any given case on which information will be sought are the following: (a) Can a perfect foot be obtained? (b) Will a shapely foot result from treatment? (c) Will the patient be able to walk comfortably and rapidly? (d) What possibility is there of relapse, and, if relapse occur, can the foot again be rectified? (e) The duration of treatment? The answer to question (a) depends upon the age at which treatment is begun and the degree of deformity. Many cases of congenital varus of the first and second degrees are cured if treatment is begun before walking is attempted. Cases of the third degree, especially after weight has been borne on the deformed feet, seldom give perfect results. In any event, persistency in treatment is essential, and it is the duty of the surgeon to insist that relapse is a likely event so long as growth is going on, and every care must be taken to prevent it. (b) A shapely foot will often result from early and continued treatment, but in some instances there will always remain a square-toed appearance and the foot is ungaily. (c) The question of comfortable walking depends upon the absence of cicatricial material after treatment. Tarsectomies and such like procedures should therefore be avoided if possible. (d) As to the possibility of relapse, there is very likelihood of it in congenital cases unless persistent care is exercised all through childhood and adolescence, but relapsed varus is capable of much improvement by the methods to be detailed subsequently. (e) The duration of time required for treatment depends on the degree of deformity and the method adopted. The slower orthopedic methods are effectual but tedious. A deformity of moderate severity can be reduced by manipulation and wrenching in two to three months.

**Diagnosis**—Some difficulty will arise in distinguishing in a child congenital and paralytic equino-varus, but the main points are, that in paralytic equino-varus the limb is wasted, cold,

and blue, and the electrical reactions are entirely lost in the affected muscles. From spastic paralysis the diagnosis of congenital equino-varus is more difficult. The presence of rigidity of the knees, adduction of the thighs, flexion of the forearm and contraction of the hand, are points sufficiently distinctive of spastic paralysis. The hysterical form of equino-varus readily disappears under an anæsthetic.

**Treatment**—Of slight cases or cases of the first degree.—The feet in this degree can be brought to a straight line with the legs by manipulation, and sometimes everted. But when the pressure is relaxed they spring back to their original position. Also they cannot be fully dorsiflexed when they are straightened and everted. Such cases can be treated by manipulation alone or by manipulation combined with massage and retention apparatus. In manipulation the movements to be practised are abduction and eversion at the transverse tarsal and sub-astragaloid joints, and flexion and extension of the whole foot of the ankle finishing up with circumduction. The movements should be easy and gradual, and should last about five to ten minutes three times a day. Manipulation can be combined with retention apparatus in the following way. A straight well-padded piece of soft iron is bent to the shape of the deformed foot and applied to the outer border of the limb. Gradually the angle of the splint is diminished until the foot can be fixed in a straight line with the leg without pain. This suffices to remove the varus. The equinus can be overcome by the use of a tin shoe with a quadrant movement at the ankle. In place of the splints many surgeons prefer to use plaster of Paris frequently changed, additional rectification being obtained whenever a fresh plaster of Paris splint is put on; this should be at least every third day.

**OF THE SECOND DEGREE**—Here the foot can neither be fully everted nor brought into a straight line with the leg. In attempting to do so the tendons of the tibialis anticus and posticus and the flexor longus pollicis, together with the tendo Achillis, become tense. Cases of this degree may be cured by (a) tenotomy with the after-use of shoes and apparatus, or (b) tenotomy followed by wrenching on two or three occasions and putting the foot in plaster of Paris after each partial correction. As this degree is usually found in children under four years of age, the bones and ligaments are still elastic, and it is not necessary to resort to deep operations such as Phelps' operation or the various kinds of tarsotomy. The following tendons need division. In the first stage, the tibialis anticus and posticus, the plantar fascia and the anterior fasciculus of the internal lateral ligament of the ankle. As to whether reposition of the front part of the foot should be aimed at immediately after tenotomy or gradually is a matter of somewhat diverse opinion. As a rule gradual

reposition by means of the flexible iron splint is to be preferred, and the varus will be gradually overcome until the foot is in a straight line with the leg. Then and only then should the tendo Achillis be divided to reduce the equinus. After the tendo Achillis has been divided an Adam's varus splint, or a tin shoe with a quadrant at the ankle, is substituted for the flexible iron splint. As in the first degree, some surgeons prefer to use plaster of Paris instead of splints or shoes, but its disadvantages are that its use involves a good deal of trouble if it is renewed sufficiently frequently, and the foot cannot be manipulated while it is on. This is a very essential part of the proceedings. After-treatment, extending over some years, by means of walking apparatus, is required. In some of these cases there is very considerable inversion of the whole limb. This may sometimes be overcome by frequent twisting movements on the part of the attendant or by linear osteotomy.

**OF THE THIRD DEGREE**—Rigid or resistant club foot. Here all the deformities are exaggerated and the bony prominences are well marked on the outer side of the foot and are the sites of considerable pain. The best form of treatment for these rigid feet is, in the writer's opinion, a gradual one. There is no course that answers so well as the following: Take the patient off his feet for two or three weeks and give him complete rest, by so doing the painful spasm of the muscles passes away and the foot soon becomes less rigid. The degree of suppleness which returns to the foot is surprising. Tenotomy of the tibial tendons and plantar fascia is now performed, and the varus is overcome by using a malleable iron splint or by employing a Scarpa's shoe. Happily successful as treatment on these lines is, the length of time occupied has induced surgeons to devise other means of overcoming the difficulties and restoring the foot. A valuable adjunct to treatment is forcible rectification or wrenching. The best apparatus is Thomas' wrench. It is better in obstinate cases to employ it frequently and moderately than with violence. The more extensive operative measures are free subcutaneous division of all the resistant structures at one sitting, Phelps' operation, tarsal osteotomy and tarsotomy. As to the value of either of these operative procedures it is well to remark that Phelps' operation or treatment by open incision has been for some time on its trial and has not given satisfaction. Free subcutaneous section is followed by so much scarring that the foot remains rigid although it may be of a good shape. Tarsal osteotomy is often insufficient because it is difficult so to plan the incisions as to allow of the bones being placed in their proper axes. Of the many forms of tarsotomy it may be said that removal of the astragalus is the best. Now, in commencing the treatment of a case, either in this degree or the next, it is important



to decide at once if the foot is capable of reposition by the gradual method, or requires tarsectomy. If the latter measure is called for, the writer has no hesitation in pronouncing astragalectomy to be the operation which is likely to yield excellent results on the following grounds—the resulting good movement of the ankle, the immediate and permanent correction of the deformity, the absence of recurrence, and the ease of the operation. As to wedge-shaped tarsectomies, the operation is easy, and it is very tempting, if a foot is too short on the inner side and too long on the outer, to saw out a piece of bone and put the foot straight. But this does not appear to be a very scientific proceeding. The cause of the trouble is in the inner segment of the longitudinal arch, and there the deformity should be rectified.

**OR THE FOURTH DEGREE**—Here the distortion is exaggerated to its utmost limit and the foot is fixed, feeling as if it were set in plaster of Paris. The decision has to be made as to between astragalectomy with free division of the resisting soft structures and amputation. The latter is very rarely called for, and many patients go on hobbling about with ulcerated feet.

*Treatment of relapsed cases* are best conducted on the lines advocated for the second and third degrees as above.

Congenital talipes calcaneus is seldom very resistant to treatment, and the same may be said of calcaneo-valgus. It is necessary to divide the contracted tendons and use the malleable non splint for reposition, suitable manipulations being carried out twice daily.

#### CLUB FOOT—ACQUIRED

*Club-foot acquired—Causes*—The most usual cause is infantile paralysis, and less frequently spastic paralysis. Rarer causes are cicatrices such as result from burns, traumatism, such as injuries to bones and fractures, severance of tendons and nerves, inflammation, such as occurs in acute osteomyelitis when the rate of growth of one bone is arrested, while in the other it is normal, "talipes debilitus," a spurious form of talipes due to contraction of the tendo Achillis, and occurring in bed-ridden patients, and lastly hysterical talipes.

*Talipes Equinus*—The most common cause is spastic paralysis, less frequently infantile paralysis. The degrees of talipes equinus are as follows—

**THE FIRST DEGREE** or right-angled contraction of the tendo Achillis.—When the knee is fully extended the heel cannot be brought into complete apposition with the ground without pain or force. The results of this slight deformity are formation of corns beneath the heads of the metatarsal bones, slight lameness and shortening of the stride, and some inversion or eversion of the foot at the ankle. Many cases of so-called talipes varus or valgus are found to be equinus

when carefully examined with the knee fully extended.

**THE SECOND DEGREE**—The heel is raised well off the ground and progression takes place on the head of the metatarsal bones, beneath which corns are found. The plantar fascia is frequently contracted in this degree.

**THE THIRD DEGREE** is an exaggerated condition of the second, and the foot is bent completely backwards. The morbid changes in the bones are such as would be expected from the more or less vertical position assumed by the foot. The plantar fascia and ligaments and posterior ligament of the ankle are contracted. So too are the tendo Achillis, the long flexors, and the peroneus longus. In paralytic feet the extensors are fatty and degenerated.

The diagnosis is simple when the affection is well marked and of the second and third degrees, but in the first degree or right-angled contraction of the tendo Achillis the affection is often overlooked. Talipes arcuatus and plantaris (pes cavus) are often due to paralysis of the interossei with some weakness of the long extensor tendons.

**TREATMENT OF TALIPES EQUINUS**—In case of the first degree or right-angled contraction, manipulation and active and passive exercises designed to stretch the tendo Achillis may be employed. A toe-elevating spring attached to an outside vertical steel support is useful. But much time and trouble may frequently be saved by dividing the tendo Achillis, taking care, however, that the tendon does not become too long. The dorsiflexion should be controlled by a "stop" at the ankle. In equinus of the second degree, section of the plantar fascia is called for, and when the sole of the foot is unfolded the tendo Achillis is divided. If the toes should be clawed, the extensor tendons may be divided opposite the heads of the metatarsal bones, at the same time that the plantar fascia is divided. Immediate after-treatment consists in the use of Scarpa's shoe or plaster of Paris, and to prevent the tendo Achillis becoming too long the ankle should be controlled by a "stop." Great attention must be paid to massage and the application of the induced current in paralytic cases. In the third degree the front part of the foot should be unfolded and the tendo Achillis divided subsequently. The wrench may be freely employed if the deformity is obstinate. Should it fail, astragalectomy is called for. In spastic cases the contracted tendo Achillis should always be divided, despite the advice sometimes given to the contrary. By doing so, much lameness is avoided and the patient walks in comfort.

*Acquired talipes calcaneus* is the result either of infantile paralysis or is due to excessive lengthening of the tendo Achillis after an operation for talipes equinus. It is a very troublesome deformity to treat. Its aspect is entirely different from that of the congenital

**calcaneus** In the acquired form the heel is dropped and forms a very distinct prominence, and the arch of the foot is much increased, and there is some contraction of the plantar fascia. In all cases where the heel is much dropped and the arch of the foot unduly concave, especially if contraction of the plantar structures have ensued, the outlook is bad. The chief difficulty consists in keeping the heel up, and this is especially so when the cause is infantile paralysis.

**Treatment.**—Mechanically, a boot may be used having an outside steel support with a toe-depressing spring. Of course a contracted plantar fascia should previously have been divided. From an operative point of view many attempts have been made to shorten the tendo Achillis, but these as a rule are not successful because the tendon is extremely thin. The best results in the treatment of acquired calcaneus are obtained from tendon transplantation (*q v*).

**Talipes calcaneo-valgus and calcaneo-varus** are usually due to infantile paralysis. In calcaneo-valgus the heel is depressed and the foot is turned outwards, while in calcaneo-varus the foot is turned inwards. Sufficient indications for treatment are given under the headings of *varus*, *valgus*, and *calcaneus*.

**Talipes Arcuatus and Plantaris or Pes Cavus.**—In these deformities there is increased concavity of the arch with a corresponding dorsal convexity. In *talipes arcuatus* the arch is increased, but the heel and the balls of the toes are in a horizontal plane. If the balls of the toes fall below the level of the heel, and the arch is at the same time increased, then the condition known as *talipes plantaris* is present. The causes are slight paralysis of the anterior muscles of the leg following diphtheria, measles, scarlet fever, chorea, infantile paralysis. They are also associated with Friedrich's disease, and some say (*e.g.* Duchenne) that they are due to paralysis of the interossei and lumbricales. The most frequent symptoms are pain in walking, increase of the arch of the foot, and corns beneath the heads of the metatarsal bones. Treatment consists in division of the plantar fascia and the after-use of a Scarpa's shoe with a single uplifting movement in the sole.

**Talipes Varus.**—The acquired form is usually due to infantile paralysis and to one variety of progressive muscular atrophy in which the peronei muscles are the earliest to be affected. With regard to the appearances and treatment of a varoid foot they are sufficiently detailed in the description given of congenital *talipes equinovarus*.

**Talipes valgus** (acquired) arises either from infantile paralysis, spastic paralysis, rickets, or as a sequel to Pott's fracture. The appearances in treatment are precisely similar to that of ordinary flat-foot.

## FLAT-FOOT

(SPURIOUS TALIPES VALGUS)

**Definition.**—Flat-foot is a deformity of the feet, often painful, characterised by abduction and eversion of the foot with loss of the arch.

Weak ankles, or valgus ankles. Weakly children, especially those suffering from rickets, frequently turn their feet over in walking, and associated with this condition, which is due to relaxed ligaments, there is some genu valgum.

**General Description and Appearance of Flat-foot.**—All the changes of the foot are due to sinking of the so-called arch, not only in the longitudinal but also in the transverse direction. The depression downwards and inwards of the head of the astragalus and the outward twist of the anterior part with extreme rotation of the foot are the immediate results. The foot is increased in length on the inner side and it is broadened. Flattening of the sole is also present, and the instep loses its roundness. The inner border is convex instead of concave, and is in contact with the ground. It is also thicker than normal. The heel appears to be shortened, the internal malleolus becomes extremely prominent in severe cases and descends downwards, inwards, and backwards, so that its tip is either in a line with or even behind that of the external malleolus. On the inner aspect of the mediotarsal joint the head of the astragalus is prominent. It sinks downwards, forwards, and inwards, and the scaphoid becomes unduly prominent. The soft tissues over the astragalus and scaphoid are often hypertrophied, and false bursae and thickened epidermis may be seen at these spots. In front of the mediotarsal joint the inner border is sloped outwards, and the great toe is frequently valgoid. The outer border is commonly shortened and often raised from the ground. In many cases varicose veins and sweating of the feet coexist, so that it is probable that flat-foot arises from defective innervation of the vessels of the legs and feet. In exceptional cases wasting of the tibiae anterior is seen.

**Degrees of the Flat-foot.**—For purposes of description there may be said to be four degrees. First degree or oncoming flat-foot.—There is noticeable some sinking of the arch when the patient stands and he is told to bear the weight fully on the foot. This sinking disappears on adduction of the foot, standing on tiptoe, and on sitting. Pain is frequently present at this stage. Second degree or pronounced flat-foot.—The arch has sunk to some considerable extent although the head of the astragalus is not touching the ground. The deformity cannot be reduced by any effort of the patient, nor can the feet be voluntarily inverted. The peronei tendons and the extensor communis digitorum are seen in relief, and there is considerable muscular spasm and pain. Third degree or

**spasmodic flat-foot**—The astragalus and scaphoid are touching the ground, the foot is very tender, and the patient can only hobble, and the deformity cannot be reduced either by the patient or by the surgeon. The peronei and extensor communis digitorum tendons are in strong relief, and the thickening of the soft tissues on the inner side is much in evidence. **Fourth degree or osseous flat-foot**—In this degree the deformity is excessive, and marked changes occurring from arthritis are met with at the medio-tarsal and other joints. In some cases it is said ankylosis takes place.

*The Etiology and Causation of Flat-foot*—In the majority of cases there are three factors involved,—adolescence, feeble health, and strain on the feet out of proportion to the muscular development. Under the heading of feeble or impaired health contributing causes are, weakness following exanthemata, acute rheumatism, and anemia. Excessive strain on the foot frequently arises from occupations involving long standing on weak feet (static flat-foot). In addition to these general factors, local conditions are concerned—for example, bunions and corns on the outer side of the foot, hallux valgus, genu valgum, shortness of one limb, high-heeled and narrow boots, gout, and injury.

*Pathology and Symptoms*—Abduction of the foot is the position of weakness, and adduction of strength and activity, for the usefulness of the foot varies with the preponderance of power of the adductor muscles. When this is lost weakness and pain ensue. In fact, flat-foot depends upon simultaneous relaxation of muscles and ligaments, probably due to vascular changes. The most marked change is in the inferior calcaneo-scapoid and the calcaneo-astragaloid ligaments. As the ankle becomes more valgoid its ligaments suffer in proportion. The superficial part of the internal lateral ligament is elongated and thinned, the plantar fascia gives way, the muscles of the calf waste, and the calf loses its roundness, the peronei are in a state of tension and often stand out like cords. The alterations in the position of the bones can be readily understood if one grasps the fact that in flat-foot the astragalus descends, and the bones in front of the medio-tarsal joint are twisted on their axes, so that the inner border of the foot is brought downwards and inwards. In many cases pain is present from the first, occasionally it is felt only after a twist or sprain of the foot. It commences as a feeling of fatigue succeeded by dull aching. This aching becomes more acute, and later assumes a sharp and intense form. The tender points in the flat-foot are well defined. They are found beneath the head of the astragalus and tuberosity of the scaphoid, also below and in front of the internal malleolus, on the dorsum of the foot, and about the bases of the first and fifth metatarsal bones. The causation of the pain is first of all stretching of

the muscles, ligaments, and fasciæ, and the acute pain is probably referable to surfaces of bone not normally in contact being brought into relationship to one another at points which are not accustomed to pressure. Swelling of the feet and local puffiness are frequently seen over the tender points, and redness from time to time dependent in a degree on the amount of standing and walking. The existence of flattening of the sole is best ascertained by taking a tracing or outline of the "tread." Alteration in the gait. In cases of some severity the gait is lumbering and awkward, the patient is splay-footed, the foot is no longer elastic, and walking is further impeded by the attendant pain. In fact the patient is wooden-footed. Loss of shape in the feet is fully described under the sub-heading of appearances. Sweating of the feet is also mentioned above, and a probable explanation has been given. Flat-feet do not become cured without treatment. As a rule the pain and disability become steadily worse, and the patient has to seek relief from the inconvenience.

*Diagnosis*—Probably there is no deformity so easily and so often overlooked as slight acquired valgus. The best method of detecting the trouble is to take a tracing of the sole of the foot. The writer has known flat-foot to be treated for rheumatism, gout, and for otitis of the bones of the tarsus.

*Treatment—General*—If an anæmia be present, iron should be given for a considerable period. When the rheumatic taint exists, salicylate of soda in subacute cases, and in chronic, iodide of potassium and tincture of guaiacum, will be found serviceable. Gonorrhœal rheumatism is very intractable. In rachitic flat-foot cod-liver oil, phosphate of iron, plenty of fresh milk and pure air, will go far to effect a cure. The relief of pain is often a pressing necessity. The simplest therapeutic measure is rest, entire and absolute.

*Local Treatment*—The measures we have at our command are rest, exercises passive and active, mechanical support, and operation. As to the treatment of the first and second degrees.—In static and rickety cases the first essential is absolute rest, and with this may be combined eversion of the foot. The patient should be told to sit on a comfortable sofa or bed "tailor-fashion."

The foot rapidly loses its spasm or pain, the arch rises and the deformity is temporarily relieved. When these occur, exercises should be carried out. Those most to be recommended are tip-toe movements. Their object is to strengthen the flexors of the toes, especially the long and short flexors of the great toe. These movements should be carried out in rhythm to the swing of a pendulum or to the beat of a metronome. After a few minutes' exercise twice a day complete rest is advisable. Passive exercises may be carried out as follows.—The nurse takes the foot and performs a combination

of extension movements at the ankle with rotation at the medio-tarsal joint inwards.

**Supports.**—In slight cases it is sufficient for the patient to wear a Thomas' boot. This consists of a wedging up of the inner edge of the sole and heel, so that it is one-fourth to one-third thicker at the inner than the outer edge. In very flaccid feet a valgus pad may be added. Numerous bandages are made, and many pads and surgical soles, but the vulcanised rubber valgus pad is the best. Whitman's brace is often of value. For this the foot should be corrected under an anæsthetic as much as possible, and a plaster cast taken of it, and the brace modelled on the plaster cast. Later on further correction should be done, and another cast taken and the brace again modelled. In inveterate cases it is necessary to carry up the leg an outside steel support and to use a valgus pad in the boot, together with a T-stripe to invert the foot. The treatment of the third degree of rigid or spasmoid flat-foot. Before anything is done the patient should be placed entirely at rest for three to four weeks, and the following measures may then be employed. Forceful rectification under an anæsthetic and retention of the foot in plaster, or the more gradual method by tenotomy, passive exercises, and the employment of a modified Scarpa's shoe. Forceful rectification is carried out either by the hand or by Thomas' wrench under an anæsthetic. The gradual method consists in the first place of rest, then division of the peronei and extensor communis digitorum, and occasionally of the tendo Achillis, of the application of a malleable iron splint, to be followed later by the use of a Scarpa's shoe. In all these cases it is essential to keep up massage of the muscles of the leg and the application of the constant current, and so soon as the foot can safely bear it tip-toe exercises may be carried out. The treatment of the fourth degree.—It is in this degree, and when wrenching under an anæsthetic has failed, that operative interference on the bones is justifiable. The measures which have been carried out are resection of the sub-astragaloid joint, extirpation of the astragalus and of the scaphoid, transplantation of the posterior part of the os calcis, and excision of a wedge from the head and neck of the astragalus. Of these six the first and last are in vogue. The first is known as Ogston's operation—resection of the astragaloscapoid joint. It is done under full antiseptic precautions, and with a chisel the cartilage and a thin layer of bone are removed from the astragalus and the scaphoid in such a way as to leave on the latter a concave surface. The bones are then pegged together with ivory, the wound closed, and the foot put up in a plaster case. Stokes' operation consists in the removal of a wedge of bone from the head and neck of the astragalus, fully adducting the foot, closing the wound, and putting it up in plaster. After

all these operations great care must be taken to suitably support the foot so that no falling of the arch occurs. The writer has seen some recurrence after operation for want of this precaution.

#### METATARSALGIA

**Definition.**—This condition is a neuralgia, often extremely acute, chiefly situated in the anterior part of the foot. All authors agree that the immediate cause is pressure on the digital nerves at the head of the metatarsal bones. According to Morton, the pain is localised at the inner space between the fourth and fifth metatarsal bones. But very often one finds that it has spread to the other digital spaces, although the pain is most severe in the neighbourhood of the fourth metatarsal bone.

**Causation.**—There are cases which show that either rheumatism or gout plays some share in the production of the disease, and in this way these diseases cause a falling of the anterior transverse arch of the foot, and it is due to this falling that the nerves become pressed upon. The immediate incidence of the disease is often due to a blow or a strain in which the weight comes more immediately on the front part of the foot. In other cases the pain comes on after long standing or walking, especially in narrow boots. In most instances some degree of flat-foot is present, and this is an important point, as the flatness of the posterior part of the foot has spread to the anterior part, and the metatarsal heads have fallen out of position in such a way that pressure is caused upon the digital nerves. Mr Robert Jones has shown that the pain in the neighbourhood of the fourth metatarsal bone is due to falling of its head, and pressure upon the communicating branch between the internal and external plantar nerves which passes beneath the head of the fourth metatarsal bone. In other cases it seems that it is not this communicating branch which is pressed upon, but the inter-digital nerves.

**Symptoms.**—The patient complains of either a dull aching pain or intense burning burning pain, beginning in the front part of the foot and radiating thence up to the leg and the thigh. The pain is such as to render movement impossible. It begins shortly after rising in the morning, and becomes worse before evening, unless the patient take his boot off and rest. In fact, a very frequent expression is that "they can get no rest until they remove their boot," and this they must do no matter where or under what circumstances they are. There is not, as a rule, redness, although in exceptional cases it is described as being present. If one feels carefully, and takes a little trouble to press the metatarsophalangeal articulations separately, one finds that pressure, especially over the fourth metatarsal bone, readily elicits the pain. It can also be produced by squeezing the foot

with the hand across the heads of the metatarsal bones. Frequently the affected foot is broader in that region than is normal, and on examining the sole one or two corns will be found, usually over the heads of the third and fourth metatarsal bones. These corns are indicative of the fact that those metatarsal heads have fallen out of their normal position. Frequently, too, there is a peculiar twist in the foot, the portion in front of the tarso-metatarsal articulation is twisted inwards, so that the base of the fifth metatarsal bone is exposed to the pressure of the boot, and the patient complains of constant pain at that spot.

**Diagnosis.**—Formerly this affection was confounded with gout or rheumatism, and vain efforts were made to treat it. It is sometimes mistaken for flat-foot, and, as we have already mentioned, flat-foot is present in many of these cases. But it is not the flatness of the posterior part of the foot which gives rise to the acute pain. It is acute neuritis of the digital nerves.

As to **Prognosis**, in severe cases the only improvement or cure that can be effected is by operation.

**Treatment.**—In all instances it is advisable to search for evidence of rheumatism or rheumatoid arthritis and gout. In slight cases the acute attacks of pain may be relieved by removing the boot and soaking the foot in hot water, and the application of the oleates of morphia and atropine may temporarily relieve the pain. If boots designed on the following plan are tried, they will be found useful. They should be narrow in the instep, so as to compress the bases of the metatarsal bones, and where the heads of the metatarsal bones are, there should be depressions hollowed out in the sole so that pressure is borne in walking not on the heads of the bones but behind them. Very often, however, one finds that this measure fails to give relief, there are then the following courses open—either to excise the nerve, or to amputate the toe, or to remove the head of the metatarsal bone around which the pain is greatest. As a rule this is the fourth, and it is astonishing to observe the good effects of this procedure. The pain immediately disappears, never to return, and the mechanism of the foot is in no wise interfered with. In fact, it is the one proceeding which gives permanent relief.

#### BOW-LEGS (CURVED TIBIA AND FIBULA)

**Causes.**—*Congenital curvature* of the legs is due to malposition *in utero*. It is usually associated with shortening of the limb and talipes equino-varus. The curve is usually anterior, and at the convexity of the curve there is frequently found a depression in the skin. This has been thought to indicate that compound fracture has occurred *in utero*. But this is not so. Treatment is generally of little value in these cases, but after childhood when the

talipes has been corrected a wedge-shaped piece of bone may be removed from the tibia. Osteitis deformans and osteomalacia give rise to curvature in the tibia and fibula as in the other long bones.

*Traumatic curvature* of the bones arises most frequently from fracture.

It is also met with after injury to epiphyses, either of the tibia or fibula. The growing power of one epiphysis is arrested, while that of the other remains unchecked, so that the longer bone is invariably bent. Such cases, however, are rather of the nature of surgical curiosities. *Syphilitic curvature* is interesting, and is more frequently a manifestation of the congenital form of disease than of the acquired. It should be carefully distinguished from *rickety curvature* by the following points. In the syphilitic form the curve is purely anterior, while in the rickety form it is antero-external or antero-internal. The syphilitic curve is usually situated at the middle of the shaft, while the rickety curve is more often in the lower third. The crest of the tibia in a syphilitic curve is smooth and rounded, and in a rickety curve sharp, while the surfaces of the tibia in the former are convex, and in the latter flat or concave. Syphilitic curvature of the tibia is best appreciated by looking at the bones from the side, when it is very striking. *Osteomalacia* and *osteitis deformans* also cause curvature of the bones of the leg, the most common cause, however, is rickets.

**Appearances.**—In rickety bow-legs the tibia is flattened from side to side, and the curve is generally most marked at the lower third. The medullary canal is often narrowed in the middle of the shaft and enlarged at the extremities. On the concave side of the curve the bone is much thickened by sub-periosteal deposit, which acts as a supporting buttress to the arch. Curvature may take place in almost any direction, but the following types are found—(a) An *external curvature* generally situated at the junction of the middle and lower third of the leg. (b) A more or less *anterior curvature* of the tibia occupying the whole length of the bone or only the upper or lower third. In these cases the heel is often raised, the foot pointed, and in walking is in a position of equino-valgus. (c) An *internal curvature* is present with flattening of the bones and the feet in a varus position. Of these three types the first is common and the third rare. Occasionally there is seen a case with an internal curve in one leg and an external curve in the other.

**Prognosis.**—In bow-legs there is always a tendency to spontaneous rectification. Thus in slight cases is often complete, but in severe cases only partial. It is therefore unwise to allow any case to pass untreated, since, if the bones are soft, slight cases may very quickly become severe.

**Treatment.**—The method depends upon

whether the bones are soft or eburnated, and upon the direction of the curve and the age and social status of the patient. If the bones are soft no operative measure is called for, and all forms of curvature except the marked anterior are amenable to mechanical treatment when the bones are soft. (1) *Constitutional treatment of rickets with local manipulation*.—This method is suitable for babies who have not yet walked, for children who are not weighty, and for those in whom the bones are not unduly soft and the curve is a general rather than a localised one. The manipulations are easily carried out by means of the nurse, and should be so done as to rectify the curvature. (2) *Constitutional treatment with mechanical support and manipulation* is adapted to the following cases:—When a curve originally slight is becoming marked; when a child is weighty and cannot be kept off its legs; when the curve is localised in one part of the bone more than another; and when the child is under four years of age and the bones are not hardened. It is not necessary to keep the child off its legs provided that the splints or mechanical apparatus are acting efficiently so as to control and diminish the size of the curve. The simplest form of apparatus is an inside wooden splint from the internal condyle to the internal malleolus for external curvature, and the reverse for an internal curvature. But when the curve is compound, i.e. when it is antero-lateral, a trough splint may be used with the angle of the trough placed posteriorly and internally so as to act as an opposing force to the antero-lateral curve. Should the curve be mainly anterior, a more elaborate apparatus must be ordered.

**OPERATIVE MEASURES.**—These are osteoclasis either manually or instrumentally, linear osteotomy, and removal of a wedge from the bone. Operative interference is called for when the bones are so hard that mechanical treatment is out of the question; in children over four years of age; in cases of severe anterior curvature, and in marked instances of lateral curvature.

With regard to the choice of operation, the majority of surgeons prefer osteotomy, but some elect to perform osteoclasis. In young children in whom the bones are not very firm manual osteoclasis is to be preferred. It is carried out by fixing the thumbs at the summit of the curve and using them as a counter-resistance to the hands placed at the extremity of the curve; by a sudden combined movement of the two hands against the thumbs the bone is quickly snapped. Care must be taken that the bone is completely broken and not merely a greenstick fracture produced. Instrumental osteoclasis is carried out by one of the numerous osteoclactors. The advantage of osteoclasis is that no open wound is produced, and the fracture is simple. The leg is afterwards put up in splints or plaster of Paris. *Osteotomy* is of two kinds,

either the linear or the wedge-shaped. The limb may be put up into plaster of Paris. The removal of a wedge is rather more difficult, and much less satisfactory for the reason that it is often difficult to completely divide the periosteum posteriorly, but wedge-shaped osteotomy is called for when marked anterior curvature is present. The wedge is best removed by the chisel. Sometimes non-union occurs after these operations, and it happens more frequently than is suspected; but considering the enormous number of osteotomies that have been performed, it is not a very serious danger.

#### GENU VALGUM, VARUM, AND RECURVATUM

**Definition.**—Genu valgum is a deformity of the lower extremity in which, if the legs are fully extended on the thighs, an angle obtuse externally exists at the knee-joint.

**Varieties.**—Rickets; static; rachitis adolescentium; traumatic, such as follows fracture of the lower end of the femur or separation of the epiphysis; inflammatory, due to osteitis about the lower end of the femur; and lastly, paralytic.

**Causation.**—Three causes are assigned—bending of the lower part of the shaft of the femur and upper part of the tibia, unequal growth of the epiphysal line, and relaxation of the joints. There can be no doubt that in rickets cases relaxation of the internal lateral ligament is the primary lesion, the internal condyle becomes prominent, and the other structures become adapted to the altered position of the limbs. The results are as follows:—Shuffling gait, contraction of the biceps tendon and of the ilio-tibial band and external lateral ligament, rotation outward of the tibia, lateral mobility of the knee-joint, and some obliquity of the pelvis, and occasionally scoliosis. Flat-foot is very frequently seen in association with genu valgum.

**Symptoms.**—When the affection is coming on the patient complains of some difficulty in rapid progression, pain and tenderness over the internal lateral ligament, and becomes very readily tired.

**Treatment.**—In the rickets form the most important thing is to treat the constitutional trouble. Now in static genu valgum there are two stages met with, relaxation of ligaments and muscles, and osseous deformities arising as the result of relaxation. These stages afford us a guide in treatment. For neither in the softened stage of the bones in rickets genu valgum, nor in the early stage of relaxed muscles and ligaments in the static variety, should an osteotomy, nor an osteoclasis, be performed. The means at our command, therefore, are general treatment, rest and local manipulation, mechanical treatment, and operative treatment. The general treatment is that for rickets. Manipulations are best carried out as follows:

—The tibia should be brought firmly inwards with the right hand, while the left hand is held firmly against the lower part of the femur. The knee-joint must be maintained at full extension while these movements are carried out. Three or four movements inward are made, and the limb is allowed gradually to come back to its original position. It is important that the child should wear splints. The simplest splints are the long wooden outside splints, with a knee-cap fitted to them, and the splints secured round the pelvis. The more costly and more efficient arrangement consists of an outside steel support from the boot on both sides, and fixed around the waist by a steel pelvic band. Most cases recover in about nine months to a year under the combined effect of rest, change of air, good feeding, manipulation and splinting. But for those that do not, operative measures are called for. These are osteotomy, osteoclasis, forcible manual rectification of the knee. Osteoclasis has been adopted of late, and appears to be likely to displace osteotomy. The object of osteoclasis is to fracture the femur just above the knee-joint, a somewhat difficult proceeding to carry out exactly. For this end various forms of osteoclasis or wrenches are used. Personally, the writer is not in favour of osteoclasis for genu valgum, he much prefers osteotomy, which is done either by Macewen's or Macdonald's method. For practical purposes the latter method is best, and the writer is accustomed to use a saw in place of a chisel. Ogston's operation, or sawing off of the internal condyle, is by no means so good an operation as Macewen's or Macdonald's, and in some cases distinct stiffness of the knee has followed sawing off of the internal condyle. After the operation the limb should be put into plaster of Paris for six weeks to two months, and subsequently it may be necessary to move the knee under an anæsthetic.

*Genu varum*. — *Definition*. — Genu varum is that condition of the legs in which a line drawn from the head of the femur to the middle of the ankle-joint falls inside the centre of the knee joint.

*Causation*. — In the majority of cases rickets is the chief cause, and genu varum is in such instances constantly found associated with curved tibia. Indeed, the so-called genu varum is not limited to the knees. There is a general outward convexity of the femur and tibia, and as the knee happens to be situated very nearly in the mid-length of the limb, it is the most prominent part of the convexity. Genu varum is also met with after operation for genu valgum as the result of over-correction. It also arises from occupation, and is met with subsequently to extension of the knee. Genu varum is seen of all degrees from slight to very considerable deformity. The nature of the affection is evident at once on looking at the patient, and

the treatment is conducted on the same lines as genu valgum. In cases in which the bones are soft, and the ligaments relaxed inside, splints with manipulation and massage are sufficient. When the bones are eburnated, osteotomy at the greatest point of curvature in the limb is necessary.

#### GENU RECURVATUM

*Definition*. — A deformity characterised by hyper-extension of the knee-joint.

*Occurrence*. — It is seen associated with other conditions, namely, congenital and paralytic club foot, rickets, deformities of one limb where an excessive strain has been put upon the sound limb, also in Charcot's disease, and as a primary condition in congenital displacement of the knee. If the deformity is a hindrance to progression, a walking apparatus with a flexion spring at the joint should be worn, or arthrodesis must be performed.

#### COXA VARA

(CURVATURE OF THE NECK OF THE FEMUR)

*Definition*. — Coxa vara is a peculiar bending of some portion of the upper part of the femur in such a way that the head of the bone sinks downwards. This bending takes place in one of two positions, either at the neck, so that the head becomes horizontal and is often twisted forwards, or the bend takes place just below the trochanter minor, so that the shaft forms here an obtuse angle.

*Causation and Pathology*. — The most usual cause is undoubtedly rickets, but there are other less well-known factors at work. Softening of the bone often occurs in adolescence, and has been ascribed to rachitis adolescentium, perhaps without sufficient reason. In boys who carry heavy weights, the neck of the femur sometimes gives. It is quite possible that some of the cases of coxa vara are due to slight inflammatory changes with softening at the epiphyseal line. The affection is more common in males than females, and is more often unilateral than bilateral.

The neck of the femur gradually yields until the head of the bone is on a level with or below the top of the great trochanter. The neck is also bent in such a way as to form a curve with the convexity backwards. It is in this class of case that the difficulty in complete flexion of the thigh is met with. In a second variety of case, namely, bending outwards of the shaft just below the trochanter, this flexion difficulty is not encountered.

*Symptoms*. — In adolescence, without apparent cause, or following slight injury, the patient begins to limp and to complain of fatigue and pain about the affected joint on exertion. Shortening of the limb is soon apparent, and is accompanied by elevation of the trochanter above Nelaton's line. The limb is sometimes

flexed to a few degrees, and is often rotated outwards. Abduction of the limb is also lessened. In fact, symptoms may be summed up as follows—Peculiar stiffness of the hip, referred to "growing" pains, the stiffness is worse on rising after sitting for a time, but is relieved by complete rest, limping, if one side is affected, waddling, if both, shortening, amounting to as much as  $1\frac{1}{2}$  inch, prominence of the trochanters, especially on flexing the thighs, displacement of the trochanter above Nelaton's line, and backwards as well, rotation outwards of the limb, and eversion of the foot, limitation of inversion and final loss of abduction, with, in an extreme case, "scissor-legged" progression and inability to walk without crutches, tilting of the pelvis and consecutive scoliosis. The signs which are absent are—Suppuration, thickening of the trochanter, tenderness on pressure, absence of the up-and-down movement on traction characteristic of congenital hip displacement.

**Diagnosis.**—It is very difficult to distinguish early coxa vara from coxitis, and the administration of an anæsthetic is often called for, but there can be no doubt that a small number of cases diagnosed as incipient coxitis prove to be coxa vara. Fracture of the neck of the femur, upper part of the shaft or separation of the epiphysis, especially if not seen until some time after the accident, may be difficult of identification. So too may congenital dislocation of a slight degree, but in the Röntgen rays we have a most valuable means at our command for diagnosis.

**Prognosis.**—Complete rest quickly relieves the pain, and the depression of the head and neck of the femur cease, and in some cases the length of the limb may be restored by traction. If the deformity is left to itself, the patient can scarcely hobble along on account of the exceedingly adduction of the limbs.

**Treatment.**—In the early stages entire rest, local massage, and passive motion in the direction of the limited movements will effect much. Failing these, complete recumbency and the employment of traction to the limb are of service. When the depression of the head has apparently reached its limit, an oblique osteotomy through the great trochanter, with the limb subsequently put up at an angle of abduction of about 25 or 30 degrees, will do much to remedy the deformity. In those cases in which the bending is in the shaft just below the trochanter minor, the wedge-shaped osteotomy is of service. The subsequent use of the cork sole will be necessary on account of the shortening if the affection is unilateral.

**Degeneracy.**—The failure to reach the normal of healthy development as seen in the loss of that degree of mental power and physical completeness which is the natural heritage of the individual. See *INSANITY*,

*ETIOLOGY OF (Degeneracy), INSANITY, ITS NATURE AND SYMPTOMS (Insane Diathesis, Degeneracy)*

**Degenerates.**—Those suffering from degeneracy, those who have lost, either in mental power or in physical development, some of their racial characters. See *HYPNOTISM (Therapeutic Uses in Various and Degenerate Children)*, *LI NACI* (Definition, "degenerates"), *PARANOID (Systematized Insanity in the Degenerate)*

**Degeneration.**—A process of decay or disintegration or transformation by which the life of the cells of an organ or tissue is interfered with so that the functions of the part are less perfectly performed or are not performed at all, strictly speaking, the cells continue to live, although in a crippled condition, and are able to perform their functions in a defective fashion or, perhaps, to perform functions of a lower order, degenerations are with difficulty separated from imitations. Some of the best known degenerations may be named amyloid (waxy or lardaceous) degeneration, atheromatous, calcareous, caseous, colloid, cystic, fatty, fibroid, fibro fatty, granular, hyaline, mucoid, paucity-matous ("cloudy swelling"), and pigmentary degeneration.

**Degeneration, Nissl's.**—The changes which occur in a nerve cell when the axon is cut, such as decrease in the chromatin of the nucleus with displacement of the latter to one side, etc. See *PHYSIOLOGY, TISSUES (Nerve, Interrelationship of Nerve)*

**Degeneration, Reaction of.**—When, in the galvanic stimulation of a muscle, separated from its nerve, the anodal closing contraction becomes much exaggerated, it is said to give the reaction of degeneration. See *PHYSIOLOGY, TISSUES (Muscle, Electotomus)*

**Dehiscence.**—Swallowing. See *ALCOHOLISM (Motor Variations, Impairment of Deglutition)*, *BRAIN, AFFECTIONS OF BLOOD-VESSELS (Paralysis from Vascular Lesions, Pseudo-hulbar Paralysis, Refraxy)*, *BRAIN, CEREBELLUM, AFFECTIONS OF (Tumors, Difficulty of Deglutition)*, *LARYNX, CHRONIC INFECTIVE DISEASES (Laryngeal Phthisis)*, *LUNGS, GANGRENE OF (Deglutition Pneumonia)*, *MUSCLES, DISEASES OF (Trichinosis, Symptoms, Implication of Pharyngeal Muscles)*, *PHYSIOLOGY, FOOD AND DIGESTION (Swallowing)*

**Dehiscence.**—Splitting open or bursting, e.g. the dehiscence of a Graafian follicle in the ovary during ovulation. See *GENERATION, FEMALE ORGANS OF (Ovary)*

**Dehumanisation.**—The loss of human characteristics (mental and physical) as seen in the insane.



**Deiters' Nucleus.**—The middle nucleus (in the pons) of the outer vestibular nucleus of the vestibular root of the eighth cranial nerve. See BRAIN, PHYSIOLOGY OF (*Cranial Nerves*), PHYSIOLOGY, NERVOUS SYSTEM (*Medulla Oblongata, Cerebellum*)

**Dejection.**—The act of emptying the bowels, or the matters evacuated from them, also, mental depression (*dejectio animi*)

**Delayed Labour.** See LABOUR, PRECIPITATE AND PROLONGED, etc

**Delhi Boil.** See FURUNCULUS ORIENTALIS

**Deligation.**—The ligation or tying of arteries

**Deliquium Animi.**—Mental failing, fainting, *delectio animi*

**Deliramentum.**—Delirium (derived probably from *de*, from, and *lira*, a furrow, and meaning, therefore, a deviation from the normal standard of mental health), the French word *délie*, it is to be noted, means mania as well as delirium, thus *délie aigue* is acute mania

**Delirants.**—General cerebral stimulants, causing mental excitement, such as belladonna, stramonium, hyoscyamus, alcohol, tea, coffee, coca, tobacco, camphor, opium, etc. Some of these drugs have first a stimulant and later a depressing or paralyzing influence, e.g. opium

**Delirious Mania.**—SYNONYMS—*Acute delirious mania, delirium acutum, Bell's mania, typhomania*

**Definition.**—An acute delirium characterised by precipitate onset, rapid course, fever, extreme prostration, and usually fatal issue

**History.**—This disease was described fifty years ago by Dr Luther Bell as "a form of disease resembling some advanced stages of mania and fever." Since then numerous cases have been reported in America, in this country, and on the continent. The term acute delirious mania has unfortunately been frequently misapplied to severe cases of mania or of excited melancholia, and in consequence some observers have been led to doubt the existence of Bell's mania as a distinct clinical entity. This would appear, however, to be a mistaken view, as there is abundant evidence of the existence of a quite sharply characterised acute delirious mania such as Bell described

**Etiology.**—Most of the cases occur between the ages of thirty and fifty. The patients are frequently of neurotic constitution, and the symptoms may follow worry, over-work, or disappointment. Other cases have arisen in association with pneumonia, septic infection, insolation, alcoholism, and injury, especially of the head

**Symptoms.**—There is usually, if not always, a prodromal stage during which the patient suffers from lassitude and depression of spirits, and often also from insomnia. The acute stage of the disease is entered on suddenly, and in the course of a few minutes the patient may pass into a state of the wildest excitement with vivid hallucinations, delusions of no fixed type, and outbreaks of great violence. He rushes about gesticulating, talking, singing, laughing, crying. He refuses food, and is entirely sleepless. This delirious stage may continue for several days, until at length he passes into a condition of extreme prostration. The movements become ataxic, and are interrupted by twitching and spasms. The talking is replaced by an unintelligible muttering. The tongue is now found to be covered with thick fur. Sordes appear upon the teeth. The pulse is quick and feeble. The temperature is found to be from four to six degrees above the normal. The whole aspect of the patient is that of profound nervous exhaustion. The appearance of this typhoid stage is very characteristic, and suggested the name typhomania originally applied to the disease by Bell.

The early excitement, the want of food, and the want of sleep result in rapid wasting of the body, which may become quite extreme under the strain of a colloquative diaphana which sometimes sets in. Perspiration is often profuse, and eruptions, pustular, bullous, or petechial, may be present. The urine is scanty, high-colored, and may be albuminous. Gradually the patient sinks into deep coma, and death takes place in from three days to three weeks from the onset of the delirium. In the rare cases that recover, the patient does not become comatose, but passes through a prolonged convalescence, regaining his bodily and mental health very slowly, and often imperfectly.

**Prognosis.**—As already stated, the disease is usually fatal, but the mortality is variously stated by different writers. When death does not occur a good deal of mental feebleness, amounting even to complete dementia, may be left behind.

**Pathology.**—The principal anatomical changes are congestion of the meninges and of the cerebral cortex, and occasionally areas of softening. Pinetate hemorrhages are present, and leucocytes and red blood-corpuscles are found in the perivascular spaces.

The disease appears to be an acute infection. Organisms have been isolated from fatal cases. Rason, for example, obtained from an area of softening in a fatal case pure cultures of a small bacillus with rounded ends which grew in the ordinary culture media at the temperature of the body, or of the room, and which was stained by the ordinary aniline dyes, but not by Gram's method. Rabbits inoculated beneath the dura died in two days, beneath the skin, in four to

six days, and in both cases cerebral congestion and oedema were found

**Diagnosis.**—The clinical history of Bell's mania differs considerably from that of ordinary acute mania or excited melancholia, though some difficulty may be experienced in diagnosis during the period of excitement. The precipitate onset, the extraordinary intensity of the symptoms, the rapid changes in the type of the delusions and hallucinations, and the fever are points of importance. Greater difficulty in diagnosis is sometimes presented by other febrile diseases associated with delirium. The delirious stage of Bell's mania is sometimes of very brief duration, and the patient quickly passes into the condition of prostration, to which the term "typhoid" has been applied. In this condition the case is readily mistaken for one of typhoid fever, but a consideration of the history, examination of the patient, and the use of Vidal's test should lead to a correct diagnosis. Acute pneumonia with delirium, and meningitis with muscular symptoms, may give rise to difficulty in diagnosis. The disease can hardly be mistaken for delirium tremens. A febrile delirium occasionally occurs in association with malarial poisoning.

**Treatment.**—The treatment of this disease cannot readily be carried out in a private house, as the patient during the early stage is excessively noisy and often violent. The patient is best kept by himself in a large, cool, darkened room, and nursed by experienced attendants. He should be kept in bed, his movements being limited by the use of a restraining sheet. Forced feeding with concentrated fluid nourishment at short intervals is essential. Some writers recommend the administration of a calomel purge at the outset. None of the hypnotic drugs seem to give very satisfactory results. Opium is considered dangerous. Hyoscyne is sometimes useful in limiting the excessive muscular activity, but must be used with great caution. Several writers strongly recommend the use of ergotin. Alcohol should not be used in the earlier stages, but may prove of great value when the pulse is failing.

In cases where recovery is taking place, careful nursing for a prolonged period is necessary, and the patient must be guarded very carefully from all excitement and fatigue, such as is entailed by too early visits from friends. After recovery the patient should, if possible, go to live quietly in the country or at the sea-side, and refrain entirely from mental work for at least a year.

**Delirium.** See also ALCOHOL (*Acute Alcoholic Intoxication*), ALCOHOLIC INSANITY (*Delirium Tremens*), ALCOHOLISM (*Delirium Tremens*), AORTA, THORACIC, ANEURYSM (*Symptoms*), BRAIN, AFFECTIONS OF BLOOD-

VESSELS (*Anæmia, Hyperæmia, Oedema*), BRAIN, INFLAMMATIONS (*Acute Encephalitis*), CHOREA (*Choreic Insanity, Acute Delirium*), DELIRAMENTUM, DELIRIOUS MANIA, DELIRIUM TREMENS, FRACTURES (*Constitutional Symptoms, Delirium*), HEART, MYOCARDIUM AND ENDOCARDIUM (*Symptomatology, Cerebral Symptoms*), HEART, MYOCARDIUM AND ENDOCARDIUM (*Pulse in Muscle Failure, Delirium Cordis*), HYSTERIA (*Convulsions, Period of Delirium*), INSANITY, PATHOLOGY OF, INSANITY, NATURE AND SYMPTOMS, LABOUR, PRECIPITATE AND PROLONGED (*Obstructed Labour*), MEASLES (*Symptoms, Delirium*), MENINGITIS, TUBERCULOUS AND POSTERIOR BASIC (*Symptoms*), MORPHINOMANIA AND ALIEN DRUG HABITS (*Paraldehyde*), PNEUMONIA, CLINICAL (*Symptoms, Treatment*), PUERPERIUM, PATHOLOGY (*Insanities, Clinical Varieties*), SENILE INSANITY (*Delirium of Collapse*), TRADES, DANGEROUS (*Lead-Poisoning*), TYPHOID FEVER (*Symptoms, Nervous System*), TYPHUS FEVER (*Period of Advance, Symptoms*)

**Delirium Grandiosum.**—Monomania of Grandeur, General Paralysis

**Delirium Mussitans.**—Muttering or quiet delirium

**Delirium Tremens.** See also DRINK and Cross References.—Delirium tremens (*mania à potu*) is essentially an insanity of short duration, occurring in the course of chronic alcoholism, and due not to the direct action of alcohol on the nervous system, but to nutritive changes brought about by usually long-continued alcoholic abuse. It is characterised not only by temporary mental alienation, but by disturbances at a lower level indicated by motor and sensory symptoms.

**SYMPTOMS.**—The onset of the attack is frequently determined by a temporary excess, or may be precipitated by the occurrence of an accident or the supervision of some acute illness such as pneumonia. The patient will almost always be found to be a confirmed tippler, although it not infrequently happens that he has seldom or never been actually drunk.

At the outset of the attack the patient often complains of sleeplessness at night. He suffers from great distaste for food, and often for a few days for drink also. His movements are noticed to be markedly tremulous. In conversation he is apt to be somewhat incoherent, and his attention is difficult to fix. The acuter symptoms frequently come on at night. He becomes extremely restless, and is the subject of a busy delirium in which he wanders about the room moving the chairs or arranging the bedclothes, talking constantly the while. He is very suspicious, and frequently looks behind the curtains or under the bed to see if anything likely to hurt him is concealed there. Visual hallucinations appear, usually of a terrifying nature,

whence the disease has acquired its popular name of the "horrors." The terror inspired may be so great as to lead him to jump out of the window or run into the street half naked. Auditory hallucinations are less constant, but are not rare.

The tongue is moist, tremulous, and covered with a thick whitish-yellow fur. The pulse is rapid and soft. The temperature is usually moderately raised.

After a few days, in a mild case, the symptoms gradually subside, and sleep and appetite return. The tremor persists for a few days longer. In severe cases the course of the disease is more protracted, the insomnia persists, the distaste for food is aggravated and may be associated with troublesome vomiting, exhaustion becomes extreme, and death may occur from heart failure. In other cases again the acute symptoms pass off, but the patient remains for a time the subject of hallucinations, or of insanity with melancholic delusions. After repeated attacks the brain becomes more and more enfeebled until a condition of dementia is reached.

**Prognosis.**—In a strong patient with a healthy constitution recovery is the rule. In all cases the strength of the patient, his history as to intemperance and previous attacks, and the severity of the attack, have to be taken into account. An attack in which the patient imagines he sees small objects such as spiders or beetles about the bed is apt, other things being equal, to be more grave than one in which he sees larger objects such as cats and dogs. Where no history of long-continued intemperance can be obtained, the presence of peripheral neuritis, indicated by extreme tenderness of the calves on deep pressure, will tell its own tale. All patients must be examined carefully for evidence of acute disease, particularly pneumonia, the symptoms of which are sometimes latent.

**Treatment.**—Delirium tremens being a self-limited disease resulting often in extreme exhaustion, the principal indications for treatment are to maintain the strength and to procure sleep. The former is best carried out by keeping the patient in bed, and administering easily assimilable nourishment—milk, strong soups, etc.—at short intervals. Every effort should be made to keep the patient in bed by persuasion, but in cases with very great excitement and violence it is better to resort to mechanical restraint than to permit prolonged struggling between the patient and his attendants.

As to procuring sleep, it has to be remembered that in a mild case sleep will tend to come naturally in the course of three or four nights, and it is not advisable to push the use of hypnotics too early. Nevertheless, in the majority of cases the early excitement can be quickly reduced, the restlessness and consequent muscular exhaustion diminished, and the coming of sleep hastened by the judicious use of hypnotic drugs,

although it cannot be claimed for them that they cut short the disease. As to the drugs to be used, a combination of chloral and bromide of soda judiciously administered and with due regard to the effect obtained will be found perfectly satisfactory in most cases. Opium was at one time far more used than at present. Most of the newer hypnotics have been extensively tried during recent years.

Alcohol should be avoided as much as possible in the treatment of delirium tremens. Still it is of value in a few severe cases with marked prostration, and especially in such cases when complicated with pneumonia.

After recovery from the immediate attack further treatment is indicated on the lines laid down in the article "Alcoholism."

**Delivery.** See LABOUR, DIAGNOSIS AND MECHANISM (*Transverse Lies, Spontaneous Delivery*), LABOUR, MANAGEMENT (*After Delivery*), LABOUR, PRECIPITATE AND PROLONGED, LABOUR, FAULTS IN THE PASSENGER, LABOUR, INJURIES TO THE GENERATIVE ORGANS, LABOUR, OPERATIONS (*Induction of Premature Labour*), MEDICINE, FORENSIC (*Delivery, Signs of*), PREGNANCY, AFFECTIONS AND COMPLICATIONS, PREGNANCY, HÆMORRHAGE (*Forced Delivery*), PUERPERIUM, PHYSIOLOGY (*Evidences of Recent Delivery*), PUERPERIUM, PHYSIOLOGY (*Management*).

**Delphine.** See AI KAI OIDS (*Delphinina*), SLAVINAGRI SEMINA.

**Delta.**—The fourth letter of the Greek alphabet ( $\Delta$ ), the capital ( $\Delta$ ) is used as a sign for the freezing-point of the urine in cryoscopy, and the small letter ( $\delta$ ) for that of the blood. See CRYOSCOPY.

**Deltoid Muscle.** See MUSCLES, TRAUMATIC AFFECTIONS (*Rupture*), SHOULDER, DISEASES AND INJURIES OF (*Paralysis of the Deltoid*).

**De lunatico inquirendo.**—An inquisition in lunacy, authority to inquire into the mental state of any one by a writ from the High Court of Chancery. See LUNACY (*Lunacy Laws, Chancery Lunatics*).

**Delusional Insanity.** See INSANITY, NATURE AND SYMPTOMS (*Types of Episodic Insanity, Delusional Stupor and Insanity*), PARANOIA. See also CHOREA (*Choreic Insanity, Delusional*), CLIMACTERIC INSANITY (*Clinical Forms, Delusional*).

**Delusions.** See ALCOHOLIC INSANITY (*Clinical Types*), CLIMACTERIC INSANITY (*Insane Delusions*), INSANITY, NATURE AND SYMPTOMS (*Systematic Delusions, Fixed and Progressive Delusions*), NERVES, MULTIPLE PERIPHERAL NEURITIS (*Symptoms, Psychical*), PARANOIA (*Progressive Systematized Insanity*).

**Dementia.**—Dementia was at one time employed almost as if it were a synonym of insanity, but now it has a more restricted meaning, signifying the morbid psychical state in which there is enfeeblement (or absence) of intellect and will. It is always acquired, and on this account the term *dementia congenita* as a synonym for idiocy is an incorrect use of the word. Several varieties are enumerated, among which may be named *dementia agitata*, *dementia affectata*, *dementia alcoholic*, *dementia apathica*, *dementia apoplectica*, *dementia choreica*, *dementia chronica*, *dementia epileptica*, *dementia paralytica*, *dementia senilis*, *dementia toxica*. These names explain themselves. See also **ADOLESCENT INSANITY** (*Primary Dementia and Dementia Attenuata*), **ALCOHOLIC INSANITY** (*Permanent Dementia*), **BRAIN TUMOURS OF** (*Diagnosis*), **CHOREA** (*Hereditary Adult Chorea, Diagnosis*), **GENERAL PARALYSIS** (*Synonymus, Dementia Paralytica*), **INSANITY**, **PATHOLOGY OF** (*Changes in Central Nerve Cells*), **INSANITY**, **NATURE AND SYMPTOMS** (*Biology of Consciousness*), **INSANITY**, **NATURE AND SYMPTOMS** (*Primary Dementia*), **MEMORY IN HEALTH AND DISEASE** (*Progressive Amnesia*), **MENTAL DEFICIENCY** (*Developmental Cases, Syphilitic*), **PARANOLIA** (*Progressive Systematised Insanity*), **SENILE INSANITY** (*Simple Dementia, Complicated Dementia*).

**Demissio Animi.**—Depression of spirits.

**Demme's Bacilli.** See SKIN, BACTERIOLOGY OF (*Bacteria identified with various Diseases, Erythema Nodosum*).

**Demodex Folliculorum.** See PARASITES (*Anthropods, Arachnoides, Acarina, Demoder*), **SCABIES OR THE ITCH** (*Other Acari, Demoder*).

**Demography.**—A word recently introduced, signifying the life conditions of communities or nations as revealed by statistics of births, marriages, deaths, prevalent diseases, etc. See VITAL STATISTICS.

**Demonomania.**—Religious melancholia in which there are delusions of possession by evil spirits, demonic possession, demonopathy. See INSANITY, NATURE AND SYMPTOMS (*Melancholia*).

**Demorphinisation.** See MORPHINOMANIA (*Treatment by withdrawal of the drug, sudden or gradual, or by substitution of another drug*).

**Demulcents.**—Substances which have a soothing, lenitive, and protective effect, such as aloe, gelatin, glycerin, honey, starch, tragacanth, and white of egg.

**Dendrites.** See PHYSIOLOGY, TISSUES (*Nerve*).—The branching processes which form

a network in close proximity to a nerve cell, they do not include the axon, but arise from the other processes of dendrons.

**Dendritic Ulcer.**—A peculiar branched (or tree-like) ulcer seen on the cornea. See CORNEA (*Bullous Affections, Dendritic Ulcer*).

## Dengue.

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See also EPIDEMIOLOGY (*Seasonal Fluctuations*).

**SYNONYMS.**—Dandy, Breakbone, Three days' fever, Abu rokah, Kulinga pepo, Scarletina rheumatica, etc.

**DEFINITION.**—A specific disease, severe but not dangerous, usually distinguished by fever of sudden onset, intense muscular and joint pains, and later a tubercloid eruption. Endemic in some countries, it usually occurs in regional epidemics, and since originally recognised (1779) has thrice assumed pandemic proportions.

**GEOGRAPHICAL DISTRIBUTION.**—Among Eastern countries, Arabia, China, and particularly India and the island countries adjoining, are subject to its visitations.

Epidemics in limited parts have occurred on all sides of Africa, except the extreme south, and in Egypt and Zanzibar especially.

In Europe its outbreaks have been confined to Spain, Greece, and Asia Minor.

In the Western Hemisphere the list includes Bermuda, the West Indies, Southern States, parts of South America, and even as far south as Tahiti. It has also reached Sydney and Brisbane in Australia.

Sporadic cases are reported by Sandwich in Egypt, and also occur in Arabia, Zanzibar, Bermuda, West and East Indies, and Honduras. In some of these the disease was apparently first introduced during an epidemic period.

**ETIOLOGY.**—In suddenness, rapid diffusion, and the enormous proportion (sometimes as great as four-fifths) attacked in a community, dengue has no equal except influenza. Again, in dependence on temperature and altitude it resembles yellow fever, which in some places it has closely preceded or followed, in the latter event reproducing, according to Smut, the features of the milder cases.

Essentially a disease of the tropics, and usually selecting there the hot season, its ordinary limits are given by Manson as 32° 47' N and 23° 23' S; yet in warm weather epidemics, always checked by winter, have extended to Southern Europe and Philadelphia.

Coast and river districts and low levels suffer far more severely than inland parts and high

altitudes, perhaps a result of freer communication, a possibility supported by the fact that towns, particularly the dirty, overcrowded parts, usually provide the starting-point and chief field for its outbreaks. Additional evidence that it is communicable is found in the numberless instances recorded of direct conveyance of infection, the particular liability of immediate attendants on the sick, and in its advance along the trade routes.

All ages and both sexes, too, seem equally susceptible, and although coloured people have occasionally suffered least, they evidently possess no immunity.

Although no organism has yet been definitely proved to be the cause, McLaughlin found in the blood of twenty patients, without exception, a micrococcus possessing unique biological characteristics, and succeeded in cultivating it in media and in blood, he was, however, unable to investigate further.

Whatever be the value of his researches, the striking resemblance in mode of spread which the foregoing facts show that it bears to other diseases of proved parasitic origin strongly suggest a like cause—an organism dependent for its highest activity upon high temperature, low levels, lack of air-space, dirt, and overcrowding. One cannot doubt its being communicable, but its occurrence in epidemic form may depend upon these other factors, a supposition favoured by the immunity enjoyed in 1871-72 by ships of the Indian Squadron in free communication with infected ports, and by the fact that, although actually introduced into some, only isolated cases occurred. Together with the short incubation (a few hours to five days), it would largely explain the chief arguments against its infectious nature, namely, the large proportion almost simultaneously attacked, dependence on local conditions, and presence of sporadic cases before epidemics.

**CLINICAL FEATURES.**—The patient is often attacked with absolute suddenness, and almost at once prostrated, but sometimes premonitions, such as malaise, painful twinges, gastric uneasiness, thirst, headache, or lassitude, precede the onset by some hours—more frequently, in Maclean's opinion, towards the end of an outbreak.

Headache, severe, perhaps paroxysmal, and associated with a sense of fullness in the skull, rapidly increasing fever, and usually severe pains, usher in the attack. The first intimation is not infrequently given by twinges in a finger or elsewhere, thence rapidly extending to other parts of the body.

Pain and sense of fullness in the eyes, which become injected and ferrety, aching in the loins, and great susceptibility to external air, though rarely definite chills or rigors, are also complained of. The skin is hot, with occasional fugitive perspirations, and the face suffused

with a deep flush, disappearing momentarily on pressure, and often attended by puffy swelling. This, which is not invariable, and is usually transient, constitutes the initial eruption, and may extend over part or most of the body. With it the buccal mucous membrane is frequently involved, reddened, congested, or even superficially ulcerated, resulting in soreness of mouth and throat. Throughout the entire illness anorexia is a prominent symptom, while gastric uneasiness, nausea, or vomiting are often present, especially after ingestion of unsuitable food.

The tongue is coated or clean, with red edges and enlarged papillæ, the bowels are usually confined, and the urine is scanty and high-colored, though rarely albuminous.

The pulse is rapid or little affected, and the respirations are hurried from pain and fever.

The temperature rapidly rises, perhaps as high as 103° to 105° F or more, but declines quickly to find it maintained above 102° being in Fayet's experience exceptional. Previous disease, such as malaria, may modify this, like other symptoms, by increasing its severity and persistence and making it more ague-like.

By giddiness, pain, and faintness the patient is soon completely prostrated, and his sufferings are increased by his extreme restlessness. No posture is comfortable, while every movement causes increased torment.

Marked nervous symptoms are rare, but convulsions or occasionally mark the onset in children, and at the extremes of life resolution may be attended by great depression. Insomnia, too, is a common feature, delirium may accompany high fever, and temporary loss of smell or taste has at times been noted.

The pains experienced may be intermittent in character and referred to muscles, bones, and joints. There is often an uneasy stiffness, unaffected by passive movement, but acutely painful on any muscular contraction, worse too after rest, and frequently combined with a sense of powerlessness. There may be neuralgic, dull, aching, or boring pain, most noticeable in the morning, sometimes metastatic, and possibly accompanied by swelling of joints.

**Second Stage.**—This first stage terminates after one to three days, gradually, or more often by crisis, marked by profuse sweating and diuresis. Bilious diarrhoea and hæmorrhages may also occur now or earlier, particularly epistaxis, which has a great effect on the headache. With fall or remission of temperature the pains abate and the patient feels well, though debilitated and perhaps subject to "reminders," such as twinges, anorexia, languor, and irritability.

**Third Stage.**—From the fourth to the eighth day of illness the terminal eruption appears, sometimes attended by slight transient return of fever, and the pains recur with, at times, greater intensity than before. This eruption is

extremely variable, and may be scarlatinal, rheumatoid, or urticarial, it is usually present to some extent. Tingling, itching, and numbness of the fingers often foreshadow its appearance on the palms—its frequent starting-point. It may begin there as small red spots, disappearing on pressure, which gradually coalesce and perhaps spread all over the body, in some cases combined with swelling.

Though generally most profuse on hands and knees, the face may be first affected, as in Christie's cases, it fades in order of appearance. A branny desquamation, often trifling, practically always follows it. This begins in about three days, and may persist for several, it is accompanied in many cases by intolerable itching.

The eruption usually disappears in twenty-four hours, though it may persist two or three days, and fever, if present, is likewise transient and very slight.

At the commencement of this stage, swelling of the cervical, inguinal, and axillary glands not uncommonly occurs, if it has not done so before. At Zanzibar the occipital glands were always involved. The already noted buccal congestion and swelling of joints may also make their appearance now, and rarely, ptyalism, orchitis, or angina.

*Varieties* occur in separate outbreaks and different localities, the cases differing in severity and in the period of incidence, relative prominence, or even existence of certain features, such as swellings of joints and glands, pains, hæmorrhages, eruptions, or the occurrence of relapses.

For instance, in Calcutta, in 1853, the characteristic pains were infrequent, and the buccal mucous membrane was prominently involved.

In mild cases, too, the course may be very short and all symptoms absent, except perhaps slight fever and eruption.

True relapses occur, sometimes due to indiscretions in diet or exposure.

Serious complications, excepting rare hyperpyrexia and mild inflammation of serous membranes, are hardly ever seen unless from previous disease.

*Sequelæ*—With desquamation the pains may vanish, but in many cases they persist in one or more joints and adjacent tissues, or perhaps return after an interval of days or weeks. They often trouble the patient for a considerable time, possess many of the features seen in the acute stage, and are specially liable to occur in elderly people. The shoulders, wrists, knees, and feet are the parts more commonly involved.

Some debility, with anæmia and anorexia, is usual after severe attacks, and insomnia, swollen glands, and furunculosis are not infrequent sequelæ.

**DIAGNOSIS** is only difficult in sporadic cases or in the presence of concurrent epidemics of influenza or yellow fever. Hamilton West demon-

strates its resemblance to mild attacks of the latter, which he considers, however, distinguished by the different course, the greater frequency of jaundice, albuminuria, and hæmorrhage, and absence of the characteristic eruption.

From influenza its distinction, at times a difficult task, is aided by infrequency of complications, the season of year, and the pain, eruption, and desquamation.

Some of these features, together with epidemic prevalence, distinguish it from rheumatism, and, aided by geographical distribution, from measles and scarlatina, while in malarious countries blood examination is valuable for doubtful sporadic cases.

**PROGNOSIS** is always favourable, unless in patients already greatly debilitated, but by its effects dengue may predispose to other illness. Charles describes a rare pernicious type, with œdema of lungs, cyanosis, coma, and hyperpyrexia, as occurring in Calcutta.

**TREATMENT**—As the disease must run its course, this must be directed towards (1) limiting its diffusion, (2) avoidance of complications and sequelæ, and (3) relief of symptoms.

As complete isolation as possible in clean, well-ventilated, and ventilated rooms, easily digestible nutritious diet, a simple diaphoretic, rest and warmth for all stages will best attain these ends, but can rarely in this disease be thoroughly carried out. Purgatives and emetics should, from the distressing movement they occasion, be given only if specially indicated.

For high temperature, sponging, or, in the rare event of hyperpyrexia, ice and quinine hypodermically should be made use of, while gastric irritability is allayed by ice to suck, effervescents, and the usual remedies. Quinine is indicated in cases complicated by malaria. An ice-cap and mustard pediluvia are of value in relieving headache, or antipyrin and phenacetin may be given. These drugs will also relieve the rheumatic pains, others recommended for the purpose being belladonna and salophen, while locally small sinapisms and liniments containing chloroform may be applied. If very obstinate, however, opium must be prescribed.

Warm baths and camphorated oil help to allay itching, while debility and anæmia demand the administration of stimulants, iron, and tonics. For the rheumatic sequelæ, tonics, salicylates, and potassium iodide, with massage and faradism, have been advocated. Complete change of climate may be necessary.

**Denidation.**—The separation of the superficial part of the uterine mucosa during menstruation, it is then supposed to break down and be thrown off. If, on the other hand, a fertilised ovum is implanted on it, it forms for the ovum a *nidus* or nest, and grows into the decidual membranes.

**Denitrification.** See MICRO-ORGANISMS (*Fermentation and Putrefaction*)

**Denman's Spontaneous Evulsion.**—A sort of natural version occurring at

above the brim of the pelvis in cases of inverse presentation, often confused with 'molar' form of spontaneous delivery. See LABOUR, DIAGNOSIS AND MECHANISM (*Transverse ex, Spontaneous Delivery*)

**Dental Caries.** See TEETH (*Dental ones*). See also NOSE, ACCESSORY SINUSES, INFLAMMATION OF (*Etiology*)

**Dental Necrosis.** See TEETH (*Dental ones*)

**Dentals.** See PHYSIOLOGY, RESPIRATION *over, Speech, Consonant Sounds, Dentals*

**Dentate Nucleus.** See PHYSIOLOGY, BRAIN SYSTEM (*Cerebellum, Structure*)

**Dentifrice.**—A substance, usually a powder (or a liquid), used for cleansing the teeth. See TEETH (*Dental Caries, Prevention*)

**Dentigerous Cyst.**—A cyst containing teeth, a dermoid cyst. See MOUTH, DISEASES OF THE JAW (*Tumours of the Jaw*)

**Dentine.** See TEETH (*Genesis of the Teeth, Nature*)

**Dentistry, Mechanical.** See TEETH (*Mechanical Dentistry*)

**Dentition.** The cutting (or eruption) of teeth, or teething, the number, character, and arrangement of the teeth, often expressed in a formula, thus—incisors  $\frac{2-2}{2-2}$ , canines  $\frac{1-1}{1-1}$ ,  
cuspidals  $\frac{2-2}{2-2}$ , molars  $\frac{3-3}{3-3}$ . See CHILDREN,

DEVELOPMENT OF (*Dentition, Symptoms, Treatment*), GASTRO-INTESTINAL DISORDERS OF INFANCY (*Ailments of Dentition*), RICKETS (*General features*), SYPHILIS (*in Children, Later Signs*). See TEETH (*Genesis of the Teeth, Eruption*)

**Denture.**—A set of artificial teeth

**Deobstruent.**—A medicine or substance which can remove obstruction by opening the natural passages or pores of the body, e.g. a cathartic or diuretic drug

**Deodorants.**—Substances which destroy foul odours or fetid effluvia, deodorisers, they are not necessarily antiseptic, but many antiseptics are also deodorants. See DISINFECTION (*Deodorants*)

**Deodorising Liquid.** See BURNETT'S LIQUID

**Depilatories.**—Agents or substances

for removing growing hairs, especially hairs growing on unusual places, e.g. on the face in women, caustic applications have been commonly used, but electrolysis is the best method, the results from the use of the X-rays have not always been satisfactory. See X-RAYS (*Hypertichiosis*)

**Depletion.**—The relieving of plethora, congestion, or the overcharged vessels of the body by medical means, formerly (especially) by bleeding

**Depopulation.**—Decrease in the population of a country either because the death-rate exceeds the birth-rate, or (less correctly) on account of active emigration. See DECLINE OF THE BIRTH-RATE

**Deposit.**—A sediment in a liquid (e.g. urine) or a substance (generally abnormal) found in some organ or tissue (e.g. uratic concretions). See URINE, PATHOLOGICAL CHANGES IN (*Urinary Sediments*)

**Depressio.** See COUCHING.—The sinking of the lens with its capsule into the vitreous humour by pressure with a needle in cases of cataract

**Depression.**—A lowered state of vitality, physiological or psychical, also the displacement inward towards the cranial cavity of a fractured cranial bone, etc

**Depressor Nerve.**—The superior cardiac branch of the vagus nerve, stimulation of the upper end (after section) causes slowing of the heart (a reflex effect through the inferior cardiac branch) and lowering of the arterial blood pressure. See PHYSIOLOGY, CIRCULATION (*Physiology of the Heart, Nervous Connections*)

**Deradelphus.**—A double monster with a single head, the trunks are fused from the neck downwards, rare in the human subject, less rare in animals

**Derbyshire Neck.**—Gottie. See THYROID GLAND, MEDICAL (*Gottie*)

**Dercum's Disease.** See OBESITY (*Adiposis Dolorosa*)

**Derencephalus.**—A variety of the teratological state known as *anencephalus*, in which there is spina bifida only in the cervical region

**Derivatives.**—Medical or therapeutic means by which (it is supposed) a diseased state (such as inflammation) may be transferred (drawn away) from one part of the body (where it is a cause of great danger) to another part (where its action is less serious), blisters, cupping, leeches, etc., are examples of derivatives.

**Dermacentor Americanus.**—A variety of tick or ixodes found occasionally in horses and oxen. See SCABIES (*Other Acari, Ixodes*)

**Dermamylasis Linearis Migrants Cestrosa.** See CREEPING ERIPTION

**Dermanyssus Avium.** See SCABIES OR THE ITCH (*Other Acari, Du Vogelmilbe*)

**Dermatalgia.**—Cutaneous neuralgia, hyperaesthesia of the skin

**Dermatauxe.**—Hypertrophy or thickening of the skin, dermatolysis

**Dermatin.**—A protective application, said to consist of salicylic acid, kaolin, starch, talc, and silicic acid

**Dermatitis.** See HERPES (*Diagnosis, Dermatitis Herpetiformis*), MICRO-ORGANISMS (*Blasomycetes Dermatitis*), NEW-BORN INFANT (*Diseases, Dermatitis Exfoliativa Neonatorum*), PELLAGRA (*Symptoms, Dermatitis*), PEMPHIGUS (*Etiology and Pathology, Diagnosis from Dermatitis Herpetiformis*), PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Impetigo Herpetiformis*), PSORIASIS (*Diagnosis from Scabious Dermatitis*), SKIN, BACTERIOLOGY OF (*Seborrhoeic Dermatitis*), SYPHILIS (*Acquired, Secondary, Affections of the Skin*)

**Dermatitis Exfoliativa Neonatorum.**—A disease of the skin occurring in the second or third week of life, characterised by excessive and general (nearly local) desquamation of the cuticle, Ritter's disease, keratolysis neonatorum. See NEW-BORN INFANT (*Diseases, Dermatitis*)

### Dermatitis Herpetiformis.

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**SYNONYMS.**—Hydroa, Dühring's disease, pemphigus pruriginosus, dermatitis multiformis, including herpes gestationis. The alternative name, Hydroa, appeals to the writer to present obvious advantages over the cumbersome title applied to this disease by Dühring, which is, however, here employed in accordance with general usage.

Dermatitis herpetiformis includes a large and somewhat inchoate group of vesicular and bullous skin eruptions, having close relationships on the one hand with pemphigus, and on the other with the bullous erythematæ. It is only within the last fifteen years that the disease, as now understood, has been generally

recognised as a separate morbid entity. Hebra certainly observed cases which he regarded as variants of pemphigus, and Kaposi maintains this view even to the present day. Bazin dimly foreshadowed the outlines of the group, while a posthumous paper by Tilbury Fox, published in the *American Archives of Dermatology*, clearly showed that that distinguished author distinctly recognised the special clinical features which characterise the disease, many typical examples of which he had accurately observed. It is, however, to Dühring that the credit is usually ascribed of finally isolating dermatitis herpetiformis from its congeners, and his admirable description, published in 1884, still holds good to a large extent and almost justifies the patronymic nomenclature frequently applied to the disease. More recently Brocq, of Paris, has published many elaborate articles on the subject, insisting especially upon the existence of two separate forms of dermatitis herpetiformis—the acute and subacute,—but these distinctions are not admitted by Dühring. Unna and many other writers have combined to create a mass of ill-digested literature, out of all proportion to the practical importance of the subject, dwelling mainly upon petty details of little scientific value, as our knowledge of the etiology of the disease—which must always be the rational basis for scientific classification and differentiation—remains virtually an unknown thing.

There is much difference of opinion as to what "types" ought to be included under dermatitis herpetiformis, more especially as to the following—(1) hydroa (vel herpes) gestationis, (2) impetigo herpetiformis, (3) hydroa vacuiforme, (4) Hallopeau's chronic pustular dermatitis in eccentrically progressive groups. Without entering into any elaborate discussion on the point, it may be said that dermatologists are now practically unanimous in accepting the first and in rejecting the three last as forms of the disease under consideration. Unna and Jannesson are strongly opposed to the inclusion of any form of skin disease which is not essentially chronic in its course, and in this view the writer fully agrees.

The "types" described by Dühring are generally accepted, and are as follows—(a) The *erythematous*, including the *urticarial* forms, (b) the *vesicular*, or commonest forms, (c) the *bullous*, (d) the *papular*, seldom extensive, (e) the *pustular*, in which pustules are present from the first, and which many would classify along with impetigo herpetiformis, and (f) the *multiform*, in which the several foregoing types co-exist. With reference to type (e) Allan Jannesson has recorded a case in the *International Atlas of Rare Skin Diseases* which presented features common to impetigo herpetiformis and dermatitis herpetiformis, forming, as he thinks, a connecting link between the two. The former disease



being now regarded as not essentially connected with pregnancy or the puerperal state, Jamieson thinks that its inclusion under dermatitis herpetiformis would tend to simplify our conceptions.

Dermatitis herpetiformis is essentially a chronic affection extending over several months or years, with relapses and recurrences at varying intervals, the skin inflammation being a superficial one, and the lesions showing a marked tendency to "herpetiform" grouping, *i.e.* for various vesicles to form closely aggregated on a common erythematous base. In different attacks the type of the disease may vary. Itching, burning, or pain is almost invariably present, the first being the commonest form of dysæsthesia, and often of intolerable intensity. All are agreed that the disease is essentially of neurotic origin, and occurs in persons of nervous temperament, while a certain proportion of its subjects become insane. Oddly enough, the general health in mild cases, or during the early stages, is often practically unimpaired.

**SYMPTOMS.**—The onset of dermatitis herpetiformis is often preceded for several days by loss of appetite and malaise, sensations of chilliness or flushing, and constipation. The temperature may be raised a degree or two before the cutaneous eruption appears, and there is often considerable preliminary itching *sine lesione*. After the eruption has appeared these symptoms usually increase in intensity, and serious constitutional symptoms may occur, especially in the bullous and pustular forms. Such cases are, however, certainly infrequent, the general health being usually but very little affected. The rash is always bilateral, and usually fairly accurately symmetrical, it may occur on any part of the body, but is most frequently observed on the flexor surfaces of the arms and wrists, on the abdomen and outer sides of the thighs. The lesions at first consist of rose-red erythematous patches or slightly raised papules, these rapidly coalesce to form plaques, which are often circular and average about half an inch in diameter. These circular patches or plaques present a raised red margin, and their centre soon becomes flattened, depressed, and of purplish colour, the appearance closely resembling that of the lesions of erythema circinatum. After a day or two vesicles or bullæ form on the spreading margin, some cases thus simulating a herpes *iris* (Hallopeau), the vesicles vary in size from a millet seed to a pea, or may even become as large as an inch in diameter. In rare cases bullæ may develop in the centre of the patches, and both bullæ and vesicles may form independently of erythema. On the other hand, the erythematous patches do not necessarily vesiculate or bullate.

While the eruption is developing very marked pruritus is almost always present, and ephemeral urticarial wheals sometimes manifest themselves

As the vesicles continue to appear, the itching is supplanted by feelings of burning, pricking, or actual pain, only relieved by the escape of the fluid contents of the lesions, this evacuation does not, as a rule, occur spontaneously, but as the result of the rubbing or scratching of the patient. The vesicular contents are usually clear and serous, but may become purulent. Contiguous vesicles tend to coalesce, forming irregular, multilocular blebs, which assume a withered, puckered appearance as they begin to disappear. Finally the erythematous base upon which the blebs originally formed may disappear, the surrounding skin showing no sign of inflammation. When the vesicles are small, and the erythematous base persists, the resemblance to herpes zoster is considerable, and many cases reported as bilateral or universal zoster are undoubtedly of this nature.

Appearances such as have been described occur in successive crops either in rapid succession or at intervals of several weeks, but in any given case lesions of various ages (wheals, erythema, papules, vesicles, bullæ, and scales or scabs) are always present simultaneously during an attack, although one or other elemental lesion usually predominates. As a rule itching is more severe in the vesicular and bullous types than in the other varieties of the disease, and in the vesicular cases the intervals of quiescence between the attacks are usually short, the duration of the disease generally very protracted. In the majority of cases only some macular pigmentation remains after the subsidence of the eruption, but in exceptional instances, observed by the writer, minute, shallow atrophic pits, herpetiform in arrangement, have remained as evidences of former attacks, facilitating diagnosis in some dubious cases.

The *bullous* type is signalled by the suddenness of development of round or irregular tense blebs with very little erythema, but minute vesicles or pustules and a few papules are generally also present. When the blebs burst or are broken they dry up, forming yellowish scabs or crusts, which drop off, leaving pigmented patches. If blebs appear in rapidly successive crops, considerable infiltration of the skin may ensue and subjective symptoms may be very troublesome. The general condition may be unaffected, but there is generally a certain amount of pyrexia with chilliness and faded tongue.

The *multiform* type is characterised by the coexistence of the several types already described, and is undoubtedly the most frequent and typical form of dermatitis herpetiformis. No further description of it seems necessary here, but a word must be said regarding *Hydroa* (vel herpes) gestationis, which by universal consent is now regarded as a form of dermatitis herpetiformis occurring either during pregnancy or the puerperium. It seldom appears before

the fourth month of pregnancy, but after that date manifests itself with gradually increasing frequency up till parturition. Fresh relapses often occur after delivery and throughout the puerperal period. In some cases seen by the writer the disease first appeared two or three days after delivery. A woman who has once had an attack of hydroa as the result of pregnancy almost always has fresh attacks, and usually of increasing intensity and of earlier occurrence, with each succeeding pregnancy. Subsequently relapses usually occur independently of pregnancy, and the case becomes identical in every way with ordinary dermatitis herpetiformis. The writer has observed one case of hydroa gestationis recurring in seven successive pregnancies; the bullous affection then became practically continuous and universal; the conjunctivæ were involved, then the mouth and œsophagus were attacked, and finally the patient died after twelve years' illness from the perforation of ulcers in the ileum, presumably of the same nature as the skin lesions.

The involvement of mucous membrane in dermatitis herpetiformis is, however, of very exceptional occurrence, in marked contrast with what obtains in the bullous erythema.

**ETIOLOGY.**—That the disease is a neurosis is indicated by the severity of the disordered sensations preceding and accompanying it, by its peculiar paroxysmal course, and by the co-existence of other well-marked nervous symptoms in many of the cases. It manifests itself frequently after some mental shock or emotion, worry, or nervous breakdown, but a considerable proportion of cases have been recorded in young adults otherwise in good health. Its occurrence as the result of pregnancy suggests its "reflex" origin, but beyond this our knowledge of the etiology of hydroa is as purely hypothetical as that of pemphigus and other bullous diseases. No relationship with gout or renal disease has been established.

**MORBID ANATOMY AND PATHOLOGY.**—In sections from a case of Jamieson's of the erythematous-vesicular type there was evidence of the origin of the vesicles in the upper papillary layer, although fully formed vesicles of a loculated character were also met with in the epidermis in close relationship to the hair follicles. Elliot found some association between the sweat ducts and vesicle formation, and located the commencement of the process in the rete and interpapillary spaces. Gilchrist, however, believes that the upper layer of the corium is the starting-point, and has found no alteration in the cells bordering on the sweat ducts. Both observers found small cell infiltration in the corium, which in Gilchrist's case was found to consist largely of eosinophile cells, and these had also penetrated to a certain extent into the epidermis. Leredde and Perrin, who first demonstrated the abundance of eosinophile cells

in the serum of the bullæ and in the blood in dermatitis herpetiformis, consider it as diagnostic and as differentiating the disease from pemphigus; this has been disputed by Neusser and many other observers, who have found excess of eosinophile cells in both the blood and serum of vesicles of eczema and pemphigus. The normal proportion of these cells is from 1 to 2 per cent of the leucocytes. In a case of Morris and Whitfield's the eosinophilia rose from 4.9 per cent of all leucocytes present in the blood at the commencement of the attack to 12 per cent when the eruption was at its height. Whitfield found from 8 to 15 per cent of eosinophiles in the blood of another case of dermatitis herpetiformis, and double this amount in one of hydroa gestationis. In a case reported by Danlos eosinophiles were present to the amount of 25 per cent in the blood and 54 per cent in the serum of the blebs. On the other hand, Drysdale showed at the Pathological Society of London specimens from the blood of a case of true pemphigus containing 60 per cent of eosinophiles, and in which the serum of the blebs was extremely rich in eosinophiles; while Peter contends that eosinophilia is a more marked feature of eczema than of any of the bullous disorders which might be classified together as pemphigus.

The occasional occurrence of associated recurrent hemo-porphyrinuria, as recorded by McCall Anderson, indicates that the underlying neurosis is probably of a paroxysmal nature.

**DIFFERENTIAL DIAGNOSIS.**—Many authorities assert that it is impossible to diagnose a first attack of dermatitis herpetiformis with positiveness, and that a firm diagnosis can only be established after watching a certain number of recurrences. This view is not endorsed by the writer's experience, although the value of the history of such cases is always very great. The similarity of the lesions to those of pemphigus and erythema multiforme may easily give rise to errors in diagnosis, especially as a history of repeated recurrences is common to all three, although in different degrees. The blebs of pemphigus characteristically arise upon skin which shows no sign of erythema, and are not intermingled with the scattered papules, vesicles, and pustules so frequently present in dermatitis herpetiformis; they are usually large and show no tendency to herpetiform grouping, while the accompanying constitutional symptoms are generally severe. Itching is a marked or even predominant feature of the disease under discussion, but is seldom present in pemphigus. The therapeutic test is undoubtedly of considerable value, as most cases of true pemphigus yield rapidly to arsenic, which has no beneficial effect in dermatitis herpetiformis. The alleged value of eosinophilia as a diagnostic point has already been referred to.

Erythema multiforme is an acute disease

running a rapid course, and usually with characteristic localisation, often also involving the buccal mucous membrane. Erythema predominates over vesication, and the lesions never exhibit herpetiform grouping.

The *Hydroa vacciniforme* of Bazin (Hutchinson's summer prurigo) occurs in young persons, and only in spring and summer, its lesions are confined to the face and other parts exposed to sunlight, and tend to diminish with advancing age, they leave indelible pitted scars.

Impetigo herpetiformis presents many striking points of distinction. The pustules of which the eruption is composed are minute, military they form little groups which spread centrifugally, itching is generally absent, the general condition is grave from the start, and death generally ensues in a few days or weeks.

Vesicating urticaria may also give rise to some doubt as to diagnosis, but it almost always occurs in children, and the concomitant phenomena readily distinguish it from dermatitis herpetiformis.

Hallopeau's chronic pustular dermatitis in excentrically progressive groups is a very rare disease, all the recorded cases having been observed in St. Louis Hospital, Paris. It appears to be a local pur modification of intense severity leading to deep involvement of the skin, which becomes much thickened and fungates. A case recorded by Wickham seems to show that this condition may supervene as an epiphenomenon in cases of Duhring's disease.

The points of differentiation from herpes or any form of "eczema" have been sufficiently dwelt upon in the preceding portions of this article.

**PROGNOSIS** — Although the general health may in many cases of dermatitis herpetiformis be but little affected, the favourable prognosis attached to the disease, and considered by Duhring as one of its salient features, is far from being entirely justified, the records of fatal cases are now by no means rare. The disease, as a rule, ceases spontaneously rather than as the result of treatment, after lasting for many months or years, or a fatal result may ensue from exhaustion or septicæmia. Generally speaking, the erythematous forms and those connected with pregnancy are the most benign, while in the bullous and pustular forms the prognosis is more grave.

**TREATMENT** — General, rather than local, treatment must be relied upon in the case of dermatitis herpetiformis. The patient's mode of life must be carefully regulated and all worry and anxiety avoided, hence probably the advantages derived from a stay in some quiet country spot or at a spa, either British or Continental. The best of these are probably Harrogate or Strathpeffer, Gastein, Kissingen, or Schintznach. The diet must be ample and nutritious, but non-stimulating. Alcohol is

certainly deleterious in most cases and tends to increase itching, the moderate use of tobacco, on the other hand, appears often to be distinctly useful. Milk is extremely valuable as a food, and if well supported should be taken in considerable quantities during the day. Constipation must be carefully guarded against, it is best combated by aperient, bitter, or sulphurous waters taken first thing in the morning, and preferably warmed. The value of arsenic has been variously estimated, Hutchinson reckons it as high, but this is not the opinion of the writer nor of the majority of dermatologists. When the eruption is in its early stages it is certainly aggravated, like many other skin affections, by arsenic, but when on the wane it may perhaps be benefited by the drug cautiously administered. Arguing by analogy it also seems probable that when administered between the attacks arsenic may tend to diminish the tendency to relapses. In similar circumstances small doses of iodide of potassium seem sometimes useful. In some cases quinine in full doses is certainly beneficial. Crocker recommends the use of full doses of belladonna, beginning with fifteen minims of the tincture, and increasing it to thirty minims three times a day. Any evidences of a gouty tendency must be treated by dieting, alkalies, and diuretics, it is in such cases that baths are of special service. To control itching no drugs are more valuable than phenacetin and antipyrin in full doses. There is no valid objection to the use of chloral and bromides to obtain sleep, but morphia is decidedly contra-indicated as tending to increase irritation. A prolonged warm bath at bedtime containing sulphate of potassium (ʒij-ʒiiv), borax (ʒij), bicarbonate of soda (ʒv), bran, linseed, or size (2 to 3 lbs), in 30 gallons of water, with or without a little liquor. Carbonic detergents often alleviates itching and procures a good night's sleep. Constant rest in bed is in itself beneficial, probably by seeming comparative uniformity of temperature.

Locally Duhring warmly recommends sulphur ointment, and his opinion is endorsed by Stephen Mackenzie, it must be vigorously rubbed into the skin, the vesicles and blebs being ruptured. Ichthyol has been warmly advocated in some quarters, internally in five-grain doses, either as capsule or pill, increased up to twenty grains or more three times daily, it is really of service. Its disagreeable odour renders it seldom tolerated in this country as an external application. It may be painted on in aqueous solution from 5 to 25 per cent, or applied as a dusting powder or ointment. The following formulæ are convenient —

B Ichthyol, gr ʷ  
 Rosacin, gr ʷ  
 Pulvis amyli  
 Magnesi carbonatis, ʒā ʒss  
 Misco Fiat pulvis

## R. Ichthyol

Camphors, 33 gr \

Olei amygdalæ dulcis, ʒj.

Adipis lane, ʒj

Misce Fiat unguentum

In the erythematous form, when few or no vesicles are present, dressings soaked in the following are useful —

## R. Resorcin, gr viij

Glycerini, ℥xx

Spiritus colonicensis, ʒij

Spiritu vin iocificati, ad ʒj

Misce Fiat lotio

The writer has found nothing more useful than weak lead, tar, carbolic or naphthol lotions, but sometimes only applications containing these drugs are more grateful to the patient.

**Dermatitis Repens.**—*Definition* —

A spreading dermatitis, usually following injuries and commencing almost exclusively in the upper extremities

There is generally a history of an injury to the skin of some part of the hand, though often the injury may be so trivial that its occurrence may have to be carefully inquired for. Vesicles or a bulla develop and rupture, resulting in complete denudation of all the upper layers of the epidermis, the surface being intensely red, and oozing a clear or turbid fluid from numerous points on the surface. The denudation extends peripherally by the epidermis at the border, being undermined and raised up by evagination, forming a sodden ragged collar which can be readily cut away, but nevertheless extension continues, and the disease may travel all up the first affected limb or stop short at any point. Less frequently it extends across the trunk to the other extremity or rarely all over the body. The parts first affected may heal slowly, leaving the skin intensely red and tender. The extension may be very slow— $\frac{1}{8}$  to  $\frac{1}{4}$  of an inch a week—and drag on for many months and even for years, or the extension may be at a much more rapid rate. The sensory symptoms are burning and tension rather than itching, and after healing great and persistent tenderness.

A closely allied condition, if not a mere variant of it, are the cases described by Hallopeau, under the title "Acrodermite continue," and by Fröche and Stowers, who acknowledge their resemblance and alliance to dermatitis repens, while Hallopeau considers them separate affections. The actual lesions of the skin on the extremities are practically identical, but Hallopeau attaches importance to the following differences. In Dermatitis repens the disease, if it extends beyond the hands, does so by direct extension of the border, while in Acrodermatitis it is by the formation of fresh foci, and the large areas are formed by their coalescence.

In acrodermatitis there is no permanent

healing of the older diseased areas, and the cases go on for years with little or no improvement. The nails are damaged and may be shed, and the oral mucous membrane may be affected. An antecedent injury has been present in some but not all of the cases.

*Pathogeny*—The theory most consonant with the clinical facts is that there is a peripheral neuritis with secondary microbial invasion of the damaged area, but Hallopeau only admits the microbial origin of acrodermatitis.

*Diagnosis*—Eczema is the disease for which dermatitis repens was mistaken until differentiated by the author. The differences are the extension at the periphery by the constant fluid exudation under the border, which forms a well-defined margin, the persistence of the lesions for long periods with slow but constant extension, and the difficulty of healing the denuded surface as well as of checking extension, and the rarity of the formation of fresh foci of disease far away from the original area. Further, for a long time the affection is limited to one extremity.

*Treatment*—The disease is very refractory to treatment, which need only be local. The most successful plan has been to cut away the undermined epidermis border and paint once a day for ten days with a 10 per cent solution of permanganate of potash. The constant application of lint soaked in lactate of lead lotion (Liq Plumbi Subacet ʒj, Lactis ʒij) has also been successful. Hallopeau paints with a strong solution of nitrate of silver, but the author has not had a good result with this, and Hallopeau has only had amelioration and never a definite cure.

**Dermatitis Traumatica et Venenata.**

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CASES of dermatitis from these causes arise from one of three circumstances—(1) From the pursuance of some employment which exposes the patient to the action of the noxious agent, (2) From the accidental exposure of the patient to some such agent, (3) Feigned eruptions.

Putting aside for one moment those substances which are such powerful irritants or corrosives as to cause a practically immediate destruction of all tissues with which they come into contact, there are some points of interest common to nearly all the irritants of the skin as regards their action in producing a dermatitis. The first peculiar point in connection with occupation or accidental dermatitis is the varying degree of susceptibility found in different

people. Thus to poisons which act with the greatest severity upon some persons others may be partially or even completely immune. White relates the case of a child of six years old who died from the effects of severe ivy poisoning produced by having his skin rubbed while wet by the hands of a boy who had been rooting up plants of the poison ivy. This case is rendered still more remarkable by the fact that the boy had previously washed his hands thoroughly, under supervision, first with hot soap and water, and afterwards with vinegar. The boy who had been working with the plants had a full and apparently permanent immunity to the poison.

Secondly, some persons who are more or less susceptible to the irritant at first may be variously affected on prolonged exposure to its action, becoming sometimes immune to its effects, or sometimes more susceptible. In the second case it is, of course, imperative that the injurious occupation be entirely given up for a time, and if after a complete recovery the patient returns to his employment, he may be attacked again either at once or after a long period of immunity. For convenience of description the forms of dermatitis may be divided into —

- (1) Corrosion of the skin with formation of slough, due to very active chemical substances.
- (2) Acute inflammations of the skin usually resembling acute eczema, but in some instances restricted to an utricular oedema.
- (3) Chronic forms of simple dermatitis, which are quite indistinguishable from chronic eczema.
- (4) More or less characteristic eruptions not resembling eczema.

I. The agents producing dermatitis of this variety are: The strong mineral acids and the caustic alkalis, and some metallic salts, such as zinc chloride, etc. The contact of such compounds with the skin is usually the result either of accident or design, since it is obviously impossible that any employment could be followed which entailed constant exposure to the action of such destructive substances. In the case of the acids the effects are usually more circumscribed than in that of the caustic alkalis, owing to the fact that they all cause a coagulation of the tissue albumins and so are more or less self-limited. In all cases the result of the action is to cause a slough surrounded by an area of intense inflammation. After separation of the slough an ulcer of varying extent and size is left, which is usually slow to heal, and has a special tendency to leave an hypertrophied scar. The reaction of the slough is naturally intensely acid where caused by the action of the strong acids, and in the case of nitric and sulphuric has a yellow colour, more pronounced in the case of the former than the latter. The slough left by the action of the caustic alkalis is of a less hard consistency, and is, of course, strongly

alkaline in reaction. The colour is usually of a dirty greyish white.

*Treatment* should be directed towards the free dilution of the poison if seen early enough, and the neutralisation with a substance of the opposite chemical reaction. After separation of the slough the resulting ulcer should be dressed with some bland ointment, such as Lister's boracic ointment.

II. The agents causing acute dermatitis of the second class may be divided into —

(a) *Animal* — Jellyfish and allied species, hairy caterpillars and stinging insects, such as hornets, wasps, bees, etc., which all cause eruptions of the utricular type, the silk in cocoon, which causes an irritable eczema-like eruption on the hands of the winders, and cantharides, which causes bullæ if in strong concentration, or a peculiar pustular eruption with hard papules if constantly repeated in weak strengths.

(b) *Vegetable* — Rhus toxicodendron, venenata, diversiloba, and vernicefera, the first three generally accidental, the last occurring in the process of the manufacture of Japanese lacquer, and occasionally in the handling of the finished article, prunella obconica, cherry laurel, aesculus, amica, juniperus sabina, staphisagria, capsicum, piper nigra, oil of croton, mustard, turpentine, and thapsia. The above list, which does not pretend to contain the name of every vegetable substance causing dermatitis occasionally, includes those that are most likely to be met with in practice. Besides these there are many members of the family Urticaceæ, as instanced by the common stinging-nettle, which produce an utricular eruption.

(c) *Chemical Compounds* — Certain aniline dyes, as found in clothing, said to be due in every case to arsenic as an impurity, but probably often due to the mordant, which may be arsenic or potassium bichromate. In this connection it may be mentioned that a very severe eruption was found on the arms of some workmen, and traced to the presence of zinc chloride in the clothing. Antimonial salts cause an acute papulo-pustular eruption, but this is not often met with now except in the case of feigned eruptions. Toilet articles and cosmetics have been not infrequently found as causes of an outbreak of eczematoid eruption, especially hair dyes which contain pyrogallolic acid or nitrate of silver. Neisser traced several cases of obstinate eczema of the lips to the use of a dentifrice containing many aromatics and some salol. Pyrogallolic acid is sometimes also the cause of eruption on the hands of photographers, but as they are often handling numerous other chemicals, it is difficult to find out exactly the cause in any given instance. Phenyl hydrazin has lately been found by a chemist experimenting with it to give rise to a very acute vesicular eruption resembling eczema.

Lastly, many substances used in surgical dressing are apt to cause acute dermatitis, such as iodoform, carbolic acid, and collodion, which last sometimes causes blisters whenever applied.

(d) *Physical Agencies* — X-rays (see "X-Rays")

The eruptions belonging to this class fall into two subdivisions, namely, the urticarial or oedematous and the true inflammatory. As types of each respectively the lesion produced by the common stinging-nettle and that produced by one of the poisonous plants, *litsea toxicodendron*, will be described.

A *The former* needs only the shortest notice, as the rash is almost always quite evanescent in character. It must not be forgotten, however, that in the case of some of the tropical plants the effects are much more lasting and severe, and that with some of the jellyfishes eruptions have occurred which, although beginning as urticaria, ended with gangrene. The symptoms of the urticarial class are then, first, shortly after the contact with the poisonous body, a circumscribed hyperemia of the irritated spot associated with sharp burning. In a few moments there occurs an exudation of serum into the hyperemic area, with the result that a pinkish swelling is produced which changes to a yellowish white as the tension in the oedematous papule is gradually raised, and the capillaries are closed by the surrounding pressure. After remaining in this state for some time, usually about half an hour, the exuded serum becomes gradually reabsorbed, and nothing remains but a slight passive hyperemia to mark the spot where the reaction occurred. If the lesion is situated on some spot where the subcutaneous tissue is very loose, such as the eyelid or scrotum, the swelling is apt to be very much greater and may completely close the eye. *Treatment* consists in the application of evaporating lotions if the pain is very severe.

B *The acute eczematoid dermatitis* commences as a local hyperemia or erythema of varying extent. Compared with the erythematous stage of so-called idiopathic eczema this traumatic erythema will be generally found to be more brilliant in colouring and more acute in onset, thus resembling erysipelas. In some cases the disease may go no farther than this, the hyperemia subsiding after a few hours, and leaving nothing behind but a slight yellowish discoloration due to diapedesis of red blood-corpuscles, to be followed by an insignificant desquamation of the damaged epithelium. More frequently, however, the hyperemia is rapidly followed by serous exudation into the corium and, later, into the epidermis itself. This is shown clinically by a marked thickening and swelling of the skin, accompanied by obliteration of the normal folds, and by a slightly translucent appearance in those situations where

the skin is thin and the subcutaneous tissue loose, such as the eyelids, penis, and scrotum.

After the exudation of serum has gone on for a certain time the fluid begins to pass upwards into the epidermis, distending the intercellular canals, rupturing the connecting prickles, and pushing aside the cells of the mucous layer so as to form small vesicular cavities. Most of the vesicles thus formed burst, either from pressure of the contained fluid or from external violence. The condition then found is one of intensely inflamed skin covered only by the moist layers of the epidermis and freely pouring out a straw-coloured fluid which dries into gummy crusts on the surface. If the skin be no further exposed to the action of the irritant the exudation of fluid gradually diminishes until it ceases altogether, a new horny layer is formed, and the surface gradually returns to the normal. Frequently the exudation into the mucous layer is so rapid as to raise the horny layer in large areas, and then bullae are the result, or in other cases where the irritant causes a marked emigration of leucocytes, the vesicles may become quite cloudy and purulent, notably so in the cases of antimonial salts and croton oil. In some cases, again, the inflammation may become so intense as to cause death of the tissues of the papillary layer, in which case ulceration will take place, and the disease can only terminate by the formation of a scar. This, it should be noted, however, is by no means a frequent occurrence in the acute eczematoid forms of dermatitis, but is much more often found in the more chronic cases of irritation by some active chemical agent. As regards the duration of the acute forms of dermatitis no exact time limit can be given. The effects of the poison may be limited to a slight and evanescent redness which passes off within a few hours, or there may be considerable exudation with vesicle formation, in which case recovery will be unlikely to be complete in less than a fortnight, or if the disease reaches a high degree of severity, even without ulceration, it will generally last several weeks.

In addition to this, one must remember that some cases of apparently simple acute traumatic dermatitis do not recover of themselves, but after some improvement has taken place tend to pass into a chronic state which is entirely indistinguishable from chronic, idiopathic eczema.

The *subjective symptoms* of acute traumatic dermatitis are in no way characteristic, but are simply those of acute inflammation of the skin, the affection usually beginning with tingling and itching, which is followed by a more or less severe burning and itching as the inflammation progresses to its height.

The *diagnosis* of acute dermatitis from acute eczema is apt to be extremely difficult or impossible. The site and history are important in every case. The sites most usually affected

are —The hands and forearms, the face, especially round the eyes, the neck, the scrotum, and the inner side of the thighs. The reason for the localisation on the hands and face is obviously the greater frequency of exposure of these parts, while probably the scrotum and thighs are often attacked from the extreme delicacy of the skin of these parts, so that any chance contact with hands covered with the irritant is almost sure to produce the eruption. The *prognosis* of the acute forms of dermatitis is almost invariably favourable, though cases have been known where death has ensued, probably from absorption of the poison through the skin, denuded as it is of its protective horny layer.

The *treatment* should be that for any extremely acute inflammation of the skin. Steps should be taken to prevent any further exposure to the influence of the irritant. If the eruption is extensive and severe the patient should be kept in the recumbent posture, so that the circulation may be rendered as quiet as possible, and only a light diet should be allowed. No internal treatment has any direct action on the course of the eruption, but it may be of advantage to give a dose of calomel at the commencement of the attack, as this will at least tend to expedite the excretion of any of the poison which may have been absorbed.

*Locally*, lotions are of most service in the early stages, such as diluted black-wash, lead, or calamine lotion. If itching is severe a very small percentage of carbolic acid may be added to the lotion. In the United States and Canada, where the disease is of much more common occurrence than in this country, owing to the greater prevalence of poisonous plants, the fluid extract of *Grindelia Robusta*, diluted one in thirty, is in great repute. After the cessation of the discharge and the more active symptoms of inflammation, recourse may be generally had with advantage to bland pastes or ointments, but in no case must any stimulating application be used unless the eruption shows signs of becoming chronic. One of the best soothing and protective applications will be found to be a cream made up of equal parts of zinc oxide, almond oil, and lime water, with fifteen grains of anhydrous lanoline to each ounce of the mixture. This application is cleanly, and is also cooling from the evaporation of the water, while it contains just sufficient grease to soften the inflamed skin and render it supple, thus obviating the uncomfortable feeling of stiffness and the liability to fissure which are apt to be present owing to the insufficiency of the horny layer.

**III CHRONIC DERMATITIS** —This is caused by the prolonged action of substances either of less virulently irritating properties or in a greater state of dilution than those which cause the acute forms of dermatitis. The offending

body may act simply *mechanically*, as in the case of fine powders, accounting for the rashes found in potters due to the fine clay, in mill stone and quarry cutters from particles of stone, in glass-paper makers, pearl cutters, knife and needle grinders, etc. Or the substance may be irritating both *mechanically and chemically*, as the finely-powdered sugar which irritates the arms of grocers, or, lastly, the irritant may act chemically only. In this last class the offending agents are almost innumerable. In contradistinction from the acute form of dermatitis the chronic form is almost confined to people whose pursuits bring them into daily contact with the offending substance. The commonest cause of all is the constant immersion of the skin in water containing an alkali, such as is present in most soaps. Another very frequent cause is the washing up of utensils which contain decomposing alcohol and weak acids, such as are found in the dregs of beer and wine glasses, hence the disease in barmen and waiters, also in cheap methylated spirit, such as is used in dissolving varnishes. Repeated contact with most metallic salts in the moist state will in time irritate the skin so as to produce a chronic dermatitis, and hence arises the disease known as galvaniser's eczema, due generally to the ammonio-nickelic oxide in the bath. Almost all the aromatic oils and perfumes are capable of inducing a chronic dermatitis when constantly used, and are generally found in face washes, powders, etc. Also many drugs are responsible for cases of skin disease in those who prepare them, instances being quinine, aconite, podophyllin, rue, vanilla, and oil of bitter orange.

Chronic dermatitis from any of these causes is usually of the dry lichenoid type. The skin is diffusely reddened and infiltrated; the epidermis is thickened generally, is of a harsh, dry character, and contains much less grease than normally. Consequently the normal folds of the skin are much deepened, and are apt to form troublesome fissures from the want of elasticity and proper cohesion of the diseased horny layer. The mouths of the follicles are often slightly gaping and hyperkeratotic, and the lanugo hairs are stunted and broken. Vesicles are found here and there from time to time, and are usually situated rather deep down in the epidermis. Secondary pyogenic infection of the fissures is very common.

The eruption is apt to spread beyond the points of actual contact with the irritant, and this fact has given rise to much discussion. Some observers hold that this spreading beyond the actual points of contact is proof that chemical irritants can call forth a true eczema, while others contend that the spreading is caused by the inoculation of the eczema virus upon the already damaged skin. Be this as it may, it is found that chronic dermatitis from external

irritants is almost invariably symmetrical, that it often lasts long after the cause has been removed, and shows little or no tendency towards spontaneous recovery, and that it is especially liable to attack the sites of predilection of idopathic eczema.

The treatment of this form of eruption is of course primarily to remove the source of irritation. After this, has been done it will be generally found necessary to apply some bland emollient preparation for some time until the more actively inflammatory symptoms have subsided and any fissures present have healed. One may then proceed cautiously with some weak form of stimulant of the class known as reducing agents, and perhaps one of the best will be found to be Pick's salicylic acid soap plaster, which should be spread rather thickly upon old coarse linen, and kept continuously applied day and night. The general principles of treatment of chronic eczema apply equally well to this artificial dermatitis, and need not be gone into in detail.

IV. The fourth class of eruptions, those that are more or less characteristic, contains only a few members.

The first group consists of those caused by arsenic, antimony, potassium bichromate, and potassium cyanide. The first three of these drugs may all of them produce an acute eczematoid dermatitis, though Richardson stated that potassium bichromate had no action on the sound skin. The feature common to them all, however, is that they are liable to produce very obstinate ulceration if they come into contact with even the slightest abrasion of the epidermis. The first three substances are all occasionally used, either in the preparation or the mordanting of aniline dyes, and potassium bichromate is also present in certain kinds of wood polishes. Potassium cyanide is used by photographers, but on account of its known poisonous action its effects upon the skin are less often seen owing to the care with which it is handled.

As regards the diagnosis of these eruptions arsenic should be suspected if there is either digestive disturbance or inflammation round the eyes, while bichromate stains the nails and skin a characteristic yellow colour. There are no special characteristics which would enable one to diagnose the presence of the other two poisons.

A very characteristic eruption is seen on the skins of tar workers, and probably the same is caused by allied substances, such as paraffins and soot, owing, in the case of the last-mentioned substance, to the traces of coal tar contained in it. The eruption appears on places where the skin is brought into contact with the offending substance, in the case of the tar worker hot anthracene oil, or more rarely creosote oil. It is greatly dependent on the habits of personal cleanliness of the individual, those who take

care to thoroughly wash off all traces of the irritant on leaving work suffering very much less than those who are careless in this respect. The sites of predilection are the face, especially the hairy parts, the backs of the hands and arms, though occasionally sparse lesions are found on the palms and the scrotum.

The earliest lesion appears to be the plugging of the follicles with misspissated tar products. The irritating action of this obstruction, probably partly mechanical and partly chemical, causes a rapid overgrowth of the cells around the follicle, so that the mouth now contains a blackened horny plug. Below this suppurative may occur, or the plug may be detached by simple mechanical movements. In either case a small depressed scar is the result, so that the arms have a honeycombed appearance, especially when viewed in an oblique light. Besides these scars, however, there are always present numerous little horny plugs which have not become expressed. The hair is usually maintained since the destruction is too superficial to affect the bulb. Associated with this stage of the eruption is also a series of red and very slightly thickened spots, which are seen under a lens to consist of dilated blood-vessels, and are, in all probability, due to the repeated hyperæmia from the splashes of the hot oils. At all events these hyperæmic spots are not acutely inflammatory since they last for years. In some cases the follicular plugs, instead of being removed by suppurization or otherwise, grow to a considerable size and then agglomerate to form the so-called tar mollusca. It is plain that there must be considerable proliferation from the first of the cells at the mouth of the follicle, in order to produce that hyperkeratosis which is one of the earliest appearances. Later this proliferation apparently affects the deeper cells, and then these grow out sideways beneath the surrounding epidermis so as to produce a hard base and a pearly edge, thus completely simulating rodent ulcer. The growth is, however, not yet malignant, though at any moment it may become so, and in most cases if left alone will slough out, leaving an ulcer which heals and produces a cribriform scar not unlike that left after vaccination. At the same time, in addition to the molluscum form of tumour, there is another, the common flat wart, the evolution of which is not quite so clear. On examining the affected skin in early stages, however, it can be seen that there is general hyperkeratosis of the parts between the follicles, though not to so marked an extent as of the follicles themselves, and it is probably from this interfollicular skin that the common wart is developed. These warts may also become the seat of malignant disease, taking on the characters of true epithelioma. It may be remarked that, in the case of sweep's cancer of the scrotum recently seen by the writer, a careful search revealed the presence of very numerous small,



flat warts on the ulnar borders of the flexor surfaces of both forearms

The *treatment* of this affection should be in the first place of a preventive kind. The men in tar works should be protected as far as possible from the splashes of the hot liquids, and should be in all cases encouraged to observe scrupulous cleanliness after cessation of work. As regards curative treatment, when the eruption has once developed there is little to be done at first. The workmen all know themselves that if they pick off the little horny projections they are liable to aggravate the disease. If any of the tumours grow to an inconvenient size it is advisable to remove them without waiting for exfoliation, and in any case a sharp look-out should be kept on all growths, so that, should any of them develop malignant tendencies, they may be removed at once.

As has been already noted, a rash strongly resembling that just described has been observed in workers with paraffin, though this agent does not appear to produce the epithelial tumours.

There is, however, another form of eruption occasionally seen on the legs of those men who habitually carry about vessels of petroleum. Usually the right leg only is affected, owing to the fact that the can is carried in the right hand. The right trouser leg gets saturated with the oil, and its action, aided perhaps by the constant friction of the skin, produces a curious bullous eruption situated on a hairy and inflamed base. There is usually a considerable amount of pyogenic infection of the eruption, which soon dies away under soothing and protective pastes.

Recently a chemical much used as a developer of photographic plates, *metol*, has been found to cause a somewhat characteristic eruption on the hands. The salient features of the eruption are—A strikingly polished appearance of the epidermis as if varnished, a diffuse, even, cyanotic hyperæmia of the affected parts, almost blue in colour, thickening of the skin itself, with the subjective symptoms of numbness, stiffness, itching, and pain. The eruption somewhat resembles both periosis and erythromelalgia. From the former it is distinguished by its occurrence in hot weather and its affecting all the fingers evenly, while from the latter it may be easily differentiated by the absence of the characteristic paroxysms of pain. Recovery takes place in two or three weeks after removal of the cause.

*Feigned Eruptions*.—Lastly, a short description must be given of the feigned eruptions. These have been separated from the main body of artificial eruptions—first, on account of the fact that they are not all true inflammations, and, secondly, because they present some peculiar features of interest. There are two classes of case to be considered—that in which the eruption is produced for obvious reasons, such as

to avoid some distasteful occupation, and that in which there is a morbid state of the mind. Patients belonging to the second class should always be most carefully watched for symptoms of mania developing. Thus in one of Shepherd's cases a girl ran out of the hospital ward, where there was no fire, with her clothes on fire.

The *types of eruption* may be divided into anomalies of secretion, such as red and black sweating, blue concretions at the mouths of the sebaceous follicles, etc., and inflammatory eruptions.

In the former class the number of colouring agents which may be used is almost unlimited, though curiously enough soot seems to be one of the most favourite applications. The pigment is generally made up with grease to make it adhere properly, so that a few drops of benzene on a tuft of cotton-wool will clean it off, leaving a perfectly normal skin beneath. The differential diagnosis of these eruptions from those of true-coloured secretion is very difficult, and is only to be accomplished either by careful watching, or in those instances in which the substance used can be identified by chemical or microscopical examination, as in a case in which extract of liquorice was used to simulate bleeding points.

In the *inflammatory* class of cases the eruption may be simply a hyperæmia, or it may be more often bullous, vesicular, and pustular, with gangrene and ulceration, or again it may be some deep-seated chronic inflammation of nodular character. Often there is, as in other kinds of hysterical manifestations, some true lesion of the skin or some eruption produced for therapeutic purposes as the forerunner of the feigned manifestation. Thus some patients who have been treated by blistering by one doctor, have afterwards procured preparations of cantharides with which to deceive people. Some of the substances used have been nitric acid, carbolic acid, cantharides, croton oil, tartar emetic, and mustard. The circumstances which should always give rise to suspicion are incongruous illeness in men, hysteria in women and girls, the age of puberty, the correspondence of the eruption with no known form of idiopathic eruption, combined with a similarity to the effects of known irritants, and the occurrence on the left side and on situations which are easily reached by the patient. In many cases the irritant will have run on the skin, producing a streak below the patch of inflammation, and the patch itself has often irregular outlines. In doubtful cases the litmus paper should always be used, as a very strong acid reaction of the tissues will remain for days when any of the mineral acids have been used. Sometimes the patch may smell of the corrosive, as in one case where the slough was produced with crude carbolic acid used for disinfecting purposes. Lastly, the area, if showing the chronic inflammatory

form, may be carefully wiped with a pledget of cotton-wool soaked in soft soap and water, and the material thus obtained tested for arsenic and antimony. Croton oil, one of the favourite applications for producing feigned eruptions, is not easily identified chemically, and its use can only be suspected by the presence of its pustular eruption on the skin and by careful search of the patient's belongings. Other methods of producing curious eruptions are by friction of the moistened skin, sometimes after previous prolonged sucking of the part, thus producing an excoriated wheal, or by the application of heat for so short a time that no true bulla formation is produced, but a superficial destruction of the epidermis with subjacent hyperæmia. Many cases of spontaneous gangrene of young women have been published on the Continent, but on reading the reports of these cases the writer is convinced that most, if not all of them, were really instances of feigned eruption.

The treatment in all doubtful cases should be the careful dressing of the affected part in such a manner that the patient shall be unable to get at the place. In such cases the eruption has often been found to occur immediately beyond the dressing, a fact which, if repeated, is practically diagnostic. Exposure of the patient, when detected, is not always satisfactory in its results, since it is usually impossible to convince the relations, and the patient only gets more sympathy. Sometimes the patient herself can be quietly lectured with better results, but it is probably always better to put her under a rigid course of treatment directed against the mental unsoundness.

## **Dermatitis Traumatica et Venenata in Coal-Miners.**

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**Introduction** — The Dermatitis Traumatica et Venenata in Coal-miners form a very interesting study to every colliery surgeon. The difficulties attendant on such an investigation are, however, numerous. The separation of the influence of general hygienic surroundings, of

poverty, of heredity, and of treatment in a more or less migratory class of workers from the direct influences exerted by the different occupations is clearly a formidable difficulty. All pit-workers do not suffer alike. Some have skins far more liable to lesions than others, so that an exciting cause in one case may have no influence in another. Again, those pitmen who are not unduly susceptible to either physical or chemical agents may show no dermatitis on the first application of an external irritant, but may do so if circumstances expose them to its influence frequently.

On the other hand, the history aids one greatly in arriving at a logical conclusion as to whether the disease is the result of occupation or not. The lesions are often quite local in their distribution and their etiology easily accounted for. Not only is this the case, but when the affection is seen only in those employed in mining, and when there is a repetition of attacks under similar circumstances, coupled with recovery when the cause is removed, one has no difficulty in stating that the disease is one due to occupation. The conclusion which one is driven to is that of the inflammation of the skin occurring in miners, and brought on by external irritants, some are due to physical and some to chemical causes.

#### **A Physical Causes Division —**

(1) *Intertrigo*. — This affection usually makes a sudden appearance between two opposed surfaces of skin. Its favourite situations are the axillæ, lower half of extensor surface of right arm and inside of lower third or lower fourth of right thigh in right-handed hewers, the left arm and left thigh in left-handed hewers, groins, scoto-femoral clefts, scrotum, perineum, natal cleft, glans penis, and prepuce. The sensations produced are those of heat and pruritus.

The history usually given is that the skin on the opposing surfaces became chafed and now feels hot and sore. The first appearance is simply a reddened surface, which, however, soon becomes raw as well as redder. On further irritation a fluid exudation covers the surface, and the result is a scalding or maceration of the affected area or areas of skin accompanied by the production of an offensive odour. The condition may end in an eczema.

The etiology of this affection comprises a number of factors. Heat, moisture, contact, pressure, movement, and friction all play their part. In addition, coal dust and coal-particles accumulate in the situations above mentioned, and by irritating the skin, especially when perspiration is practically dropping off the skin, cause an intertrigo. Another factor is the want of cleanliness, especially in the region of the genitals. In their daily ablutions some miners omit the latter region or only cleanse it partially. Again, the right-handed hewer who works with the back of his right elbow and the lower half

of the extensor surface of his right arm against the inner surface of the lower third or lower fourth of the right thigh produces an intertrigo of the parts in contact. In no case, however, have I seen a malignant condition result such as one gets in sweeps.

The treatment which has been found to be most serviceable consists in warm local boric acid baths followed by the application of carbolic oil (1 in 30), combined with rest of the affected parts. Strips of dry boric lint should be placed in the diseased clefts after each application of carbolic oil, or they may be soaked in the oil previous to their being applied. Oxide of zinc, starch, bismuth, calamine, fuller's earth, and other powders of the same nature, or combinations of these so-called harmless powders, do far more harm than good, and greatly encourage a relapse through their tendency to cake.

(2) *Eczema of the External Auditory Meatus*.—Eczema in this region is usually of the acute vesicular type, and is to be met with in all degrees of severity. The chief cause in coal-miners is the irritation produced by the presence of coal-dust and stone-particles which gain access to the external auditory meatus while the hewer is working, as he often has to do, with his head abducted. A right-handed hewer would thus have the left ear affected, a left-handed hewer the right ear. The sharp and angular particles of stone are more apt to cause irritation than the particles of coal. If the cerumen is abundant, the particles become entangled in it, and the two ultimately form a plug of impacted cerumen. When removed, they are seen to be usually tubular, and they vary in length from  $\frac{1}{2}$  to  $\frac{3}{4}$  of an inch. Such a condition causes an impairment of hearing, and gives encouragement from its recurrence to an attack of eczema. In some of the occupations in the pits the danger to life is increased by an auditory apparatus in bad working order, and hence pitmen soon consult a medical man if there be any sign of deafness. Should the eczema cause a narrowing or tortuosity of the canal throughout its whole extent, it may necessitate the pitman's changing his occupation.

Painting the affected region with friar's balsam seems to fail in curing this troublesome affection. To soften the plug thick castor oil should be dropped into the ear nightly for three nights in succession. On the fourth night the ear-channel should be syringed out with warm, weak bicarbonate of soda solution, and the plug extracted by a Volkmann's spoon or small forceps if need be. The ear is then plugged with narrow strips of lint which have been previously saturated in melted mild antiseptic ointment. The meatus soon returns to a normal condition. If eczema of the ear has already developed, syringing with warm, weak soda solution eases the pain, and this, when performed every second or third night, may in

itself be sufficient to cure the eczema. It not, the solution should be mopped up after syringing, and boric acid in fine powder blown into the ear. Salicylate of soda solution (1 in 50) is useful in allaying the pain also.

(3) *Circumscribed Inflammation of the External Auditory Meatus*.—This affection usually shows itself in the form of small boils. It is accompanied by a good deal of pain, the patient is usually "run down" in condition, and hence requires systemic as well as local treatment. The latter consists in incision of boils and syringing with warm, weak boric solution. Strips of lint soaked in carbolic oil (1 in 40) are very soothing as well as healing.

(4) *Sweat Rash*.—These are common, and are usually of an erythematous type, often scarlatiniform. A strong dose of calomel usually has the best effect.

(5) *Callosities*.—(a) *Onsetters' Hands*.—This is a rare and peculiar condition resembling Dupuytren's contraction. Coal is removed from the place where it is hewn to the pit-mouth in tubs. Youthful, called onsetters, have to push and pull these tubs, which are simply small railway waggons capable of holding, say, six hundredweights of coal. They do so by grasping the upper rim of the tub with the hands in a position of semiflexion. The movements of the hands cause oft-repeated pressure on the palms and on the flexor surfaces of the digits, with the result that the skin gets thickened and callosities form on the areas exposed to pressure. No pain is experienced, but the retraction of the fascial structures is slowly progressive, and results in a varying degree of flexion of the digits. The middle finger suffers most, and hence differs from Dupuytren's contraction, in which the middle finger is not so much flexed as the two inner ones. The condition is often bilateral. Hot baths nightly, followed by energeticunction with fatty substances, or the application of stimulating liniments, entirely fail to make any lasting impression except at a very early stage. Subcutaneous division of the contracted structures seems to be the only real remedy.

(b) *Yard-Stick Callosities*.—Officials carry yard-sticks occasionally whilst traversing the low passages of the pit, and callosities sometimes result from the grasp taken. Such a condition might prove useful in the identification of officials found dead. In one instance brought to my notice by Dr Trotter of Bedlington the official obtained support from his yard-stick while walking in a stooping position by grasping his wand about the junction of its upper and middle thirds in such a manner that two callosities were produced. By fully flexing the little finger of the right hand on the palm, and opposing the thumb to the other three the yard-stick was grasped in such a way that a callosity developed on the extensor aspect of the proximal

phalanx of the little finger, and another on the flexor and inner surfaces of the base of the thumb.

(c) *Pick-Shaft Callosities*—Every hewer shows a number of callosities on both hands produced by the constant grasping of the pick-shaft whilst working. These vary in position according as the hewer is left- or right-handed, but only to a slight degree, as the miner may have to use his left hand most the one day and the right the next.

#### B Chemical Causes Division—

(1) *Creosote Rash*—Props of wood are used to support the roof of the mine, and the process of placing these in their positions is called "timbering." The props themselves are soaked in preservative solution or solutions to protect them from fungi and moisture in the pit. I am unable to give the composition of any of the preserving fluids—the formulae seem to be trade secrets, but since the preparations smell vigorously of creosote, the rash is here termed the *Creosote Rash*. Miners are afraid of handling too many pickled props, which are black-stained and used for the dampest parts of the pit. One meets, with the creosote rash usually in adolescents who have been engaged in handling creosoted logs for several days in succession. The sites of predilection are the hands and wrists, face and neck. The rash makes its appearance in the form of a large number of small papules about the size of a pin's head, but gradually passes from this erythematous condition through a vesicular stage into a pustular one. The pustules tend to burst, and the condition assumes the appearance of a pustular eczema. The dark-brown staining material contained in the preserving fluid may assist in the production of the creosote rash. As a complication of this affection I may mention conjunctivitis, both simple and purulent. This is produced by the pitman rubbing his eyes with his unwashed hands while at work or before he takes a bath. The treatment which yields the best results consists in bathing the affected parts with a solution of salicylate of soda (1 in 50) for fifteen minutes every morning, and following this up with a liberal application of borio acid ointment—the whole to be repeated at bedtime. This is assisted by a general tonic or salicylate of soda internally, the affected parts being kept at rest as much as possible.

The general symptoms accompanying the rash are briefly as follows—Shivers, loss of appetite, headache, backache, malaise, and sickness. Papular rash appears when temperature is about 101° F. Temperature falls to about 99.5° F. when vesicular rash exhibits itself, and rises to 102.5° F. or higher when the vesicles become pustular. It then gradually falls to the extent of one degree daily until it reaches normal. The skin shows no sign of pitting, and the vesicles are not umbilicated.

(2) *Water-Rash*.—This eruption may be present on any part of the body, although it favours the hands, forearms and arms, face and neck, and the feet and legs. It occurs in those who do not handle props as well as in those who do, and simulates the creosote rash in many of its symptoms. It is apparently caused by the water in the pit, either by its dripping on the pitman whilst at work, or by his getting wet with water lying on the floor of the mine. The whole course of the disease, the rash especially, simulates smallpox. The rash is first papular, then vesicular, and finally pustular. Some of the pustules show distinct umbilication. The systemic disturbance is also similar to that of an infectious disease. Pit-water in percolating into the pit dissolves many irritating materials. In addition there is, comparatively speaking, a good deal of sulphuretted hydrogen and sulphur dioxide in the air of mines, and these when dissolved in the water may act as factors of causation, just as in the case of Delhi boils. The treatment consists in administering a good diaphoretic and diuretic mixture regularly. The diet should be light and non-stimulating, and the patient should be confined to bed. This rash seems to be a forerunner of the eczema of the feet and legs which one occasionally meets with in old miners.

(3) *Eczema of the Upper and Lower Extremities*.—This is an aggravating condition to cure. When fully developed it attacks both hands and wrists as well as the dorsum of each foot, and it may be that even the legs are included. The skin peels off in large flakes from the palms of the hands, and fissures usually extending down to the true skin make their unwelcome appearance. Both hands suffer equally. The condition is a very serious one to the miner, causing him much pain and anxiety through loss of working-time. Undoubtedly one can remove the disease by removing the cause in time, but pitmen show no delight in changing their occupation unless really compelled to do so from the severity of the skin lesion. One must therefore adopt the principle of trying to avoid depriving the skin of its natural lubricant, and to supply a substitute where the lubricating material is deficient. The handling of props ought to be discontinued therefore, and carbolic oil (1 in 40) should be energetically rubbed in night and morning. Lead and opium lotion gives relief, as also an ointment consisting of ammoniated mercury and oxide of zinc. Despite all kinds of treatment, however, the disease may remain perfectly incurable.

**Dermatobia.**—A bot-fly, found in Central America, which deposits its eggs in the skin and causes boil-like swellings (*Cutaneous myiasis*).

**Dermatol.**—Subgallate of bismuth, a dusting powder and an antiseptic; used also in diarrhoea.

**Dermatology.**—The department of medicine dealing with the diseases of the skin.

**Dermatolysis.**—Abnormal extensibility of the skin due to an alteration in its contractile faculty, also extensibility with a certain degree of hypertrophy affecting various elements of the skin and subcutaneous tissues, leading to the localised production of hanging or loose folds of skin, cutis laxa, cutis pendula, "elastic-skinned men." See PREGNANCY, INTRA-UTERINE (Diseases of the Subcutaneous Tissue)

**Dermatomycosis.**—A cutaneous affection due to the growth of a vegetable parasite such as dermatomycosis furfuracea or tinea versicolor (due to growth of *microsporon furfur*) See SKIN, PARASITES (Tinea)

**Dermatomyositis.**—An inflammatory disease of the muscles associated with edema and erythema, polymyositis See MUSCLES, DISEASES (Inflammatory)

**Dermatoses.**—A general term applied to all skin disease, and serving as the basis of the nomenclature and classification of dermatology, angioneurotic dermatoses, hemorrhagic dermatoses, and neurotic dermatoses, etc., have been described See SKIN, DISEASES, PARASITES, DRUG Eruptions, etc.

**Dermatospasmus.**—Cutis asserina See CUTIS

**Dermographia or Dermographism.**—The condition of the skin in which the stroke of the finger-nail or of the point of a pencil will raise a linear wheal, making it possible to write a word or two on the patient's back or chest See URTICARIA (Vasities, Urticaria Punctata), HÆMIEIA (Disorders of Circulation and Trophic Disorders)

**Dermoid Cysts and Tumours.** See BRAIN, SURGERY OF (Cephalocele, Diagnosis), CONJUNCTIVA, DISEASES OF (Congenital Anomalies, Dermoid Tumours), EMBRYOMATA, EYELIDS, AFFECTIONS OF (Congenital Defects, Dermoid Cysts), FALLOPIAN TUBES (Tumours, Primary Dermoid), LACRIMAL APPARATUS, DISEASES OF (Diseases of Lacrimal Sac and Nasal Duct, Dermoid Tumours), LABOUR, PRECIPITATE AND PROLONGED (Faults in the Soft Passages, Ovarian Dermoid), MEDIASTINUM (Tumours, Dermoid Cysts), MOUTH, DISEASES OF (Diseases of Floor of Mouth, Dermoid Cysts), ORBIT, DISEASES OF (Tumours, Cystic, Dermoids), OVARIES, DISEASES OF (Tumours, Dermoid Cysts), OVARIES, DISEASES OF (Primary Dermoid of Pelvic Connective Tissue), PALATE (Tumours, Dermoids), PERITONEUM, TUMOURS OF (Dermoids), SCROTUM AND TESTICLE, DISEASES OF (Tumours of Scrotum, Segregation Dermoids), TONGUE (Tumours, Cysts, Dermoid), TUMOURS (Dermoids and Teratomata)

**Dermoidectomy.**—Excision of a dermoid cyst or tumour

**Dermol.**—Chrysophanate of bi-muth

**Dermotylosis.**—Hardening or induration of the skin

**Derodidymus or Derodymus.**—A monstrosity (from Gr *derm*, neck, and *didymos*, double) with two heads and a single trunk (with two vertical columns), and two arms and two legs, and perhaps the rudiment of a third, *decaphtus diphrotipus*

**Deromelus.**—A parasitic monstrosity (from Gr *derm*, neck, and *melos*, limb) in which a limb sprouts from the region of the neck, a tracheal parasite or aucheno-melus

**Desalination.**—The removal of saline substances from the blood (e.g. in cholera).

**Desault's Splint.**—An apparatus (long outer and inner splints, and an anterior splint) used in fractures of the thigh, so called after the French surgeon, Desault (1744-1795)

**Descemet's Membrane.**—The posterior elastic lamina of the cornea, inflammation affecting it is called Descemetitis, Descemet was a French physician (1732-1810) See CORNEA (Introduction), IRIS AND CILIARY BODY (Anatomy)

**Descensus.**—Descent, e.g. of the testicles in foetal life, prolapse, e.g. of the uterus See SCROTUM AND TESTICLE, DISEASES OR (Development, Descent of Testicle), PELVIS, PERINEUM AND PELVIC FLOOR (Prolapsus Uteri).

**Desiccation.**—The action of drying up or depriving of moisture. A desiccator is an apparatus for the drying of fruit, milk, etc., powerful dehydrating agents are concentrated sulphuric acid or fused calcium chloride

**Desma- or Desmo-.**—In compound words *desma-* or *desmo-* (from Gr *derma* or *derma*, a ligament or band) means relating to a band, bandage, or ligament, or to any connecting structure. Thus *desmitic* is a connective-tissue cell, *desmatique* signifies pain in a ligament, *desmetosis* is stretching of a ligament, *desmography* or *desmology* means the description of the ligaments, *desmoid tumour* is a fibroid, *desmoma* is a tumour of the connective-tissue type, *desmosis* is a disease of the connective tissue, especially of that of the skin, and *desmurgia* is the surgical treatment of diseases or injuries by bandages

**Desmo-bacteria.**—Rod or thread-like micro-organisms, in contrast to the spherobacteria or cocci (round organisms), in Cohn's classification

**Desquamation.**—The separation or exfoliation of the epidermis, either in membranous pieces (*desquamatio membranacea*) or in small particles (*desquamatio furfuracea*), or as a complete portion or sheath (*desquamatio strigosa*). See MEASLES (*Symptoms, Eruption*), NEW-BORN INFANT (*Dermatitis Exfoliativa*), RUBELLA, ROSEOLARI (*Symptomatology, Desquamation*), SCARLET FEVER (*Symptomatology, Desquamation*), SMALLPOX (*Symptoms, Eruption*), TYPHOID FEVER (*Complications and Sequelæ, Cutaneous System*)

**Desquamative Nephritis.**—Renal inflammation in which the epithelium of the tubules of the kidneys is extensively shed

**Destructor.**—An apparatus for the efficient and cleanly disposal of refuse by means of heat, either the slow combustion furnace (e.g. Fryer's) or the high temperature or forced draught furnace (e.g. Horsfall's) may be used, there may be also a fume cremator for consuming the fumes, from the resulting clinker concrete may be obtained for making roads, mortar, etc

**Detachment of Placenta.** See LABOUR, STAGES AND DURATION (*Third Stage, Phenomena*)

**Detachment of Retina.** See RETINA AND OPTIC NERVE (*Retina, Detachment*)

**Detentio.**—Cataplexy

**Detergents.**—Cleansing substances, especially such as remove dirt, discharge, and desquamated epidermic scales from the skin or from foul ulcers by their external use, examples are found in soap and warm water, alcohol, vinegar, charcoal, pumice-stone, and sand

**Determination.**—The flow of the blood (and of other bodily fluids) to a special part, leading to congestion of that part, active hyperæmia

**Determination of Sex.**—The artificial fixing of the sex of the offspring before birth is still an unsolved problem, it does not seem that it can be done by altering the food given to the female parent during pregnancy, the general tendency of modern investigations is to show that the sex is determined at an earlier date in antenatal life than was supposed, either at the moment when the spermatozoon penetrates the ovum or in the ovum itself before impregnation, perhaps at the moment of maturation, the medical man cannot yet act as "the arbiter of the sex of the infants yet unborn"

**Detrusor.**—Literally a thruster-out or propeller (from Latin *detrudo*, I thrust away), is the name given to the muscular coat of the bladder which by its contraction expels the urine (*detrusor urinae*)

**Deutero.**—In compound words *deutero* (from Greek *δεύτερος*, second) signifies secondary.

**Deutero - albumose.** See URINE, PATHOLOGICAL CHANGES IN (*Detection of Albumoses in Urine*).

**Deuteropathic Insanity.**—Insanity caused by morbid states of other organs than the brain, secondary insanity.

**Deutero - proteoses.**—Proteids with a less complex molecule than the albumins and globulins have, and more nearly allied to the peptones than to the original proteids (The *proto-proteoses*, on the other hand, are more nearly allied to the original proteids than to the peptones) See PHYSIOLOGY, PROTOPLASM (*Classification of the Proteids*), PHYSIOLOGY, FOOD AND DIGESTION (*Stomach, Digestion, Proteolytic Period*), PHYSIOLOGY, FOOD AND DIGESTION (*Intestinal Digestion, Pancreatic*).

**Deutoplasm or Deuteroplasm.**

—The food yolk of the ovum, i.e. of the micro-blastic ovum, the deutoplasm of the hen's egg consists of white and yellow yolk, the nutritive yolk in contradistinction to the protoplasm or formative yolk

**Development.**—The series of changes by which an apparently simple structure (e.g. the ovum) becomes a highly complex organism (e.g. the embryo and fetus), also the gradual elaboration of structure and function believed to occur in and to account for the evolution of races of animals and plants, "creation belongs to eternity and development to time" (Stewart and Tait) See CHILDREN, DEVELOPMENT OF, EMBRYOLOGY, FÆTUS AND OVUM, DEVELOPMENT OF, GENERATION, FEMALE ORGANS OF (*Arrested Developments*), HEART, PHYSIOLOGY OF (*Embryology*), PALATE (*Congenital Malformations of Mouth, Development*), PHYSIOLOGY, REPRODUCTION (*Development*), PREGNANCY, PHYSIOLOGY, PREGNANCY, MULTIPLE (*Twins*), SCROTUM AND TESTICLE (*Abnormalities*), SKIN, ANATOMY AND PHYSIOLOGY (*Skin, Nails*), TEETH (*Development*), TERATOLOGY, UTERUS, MALFORMATIONS OF (*Development of Genito-Urinary Organs*)

**Developmental Idiotcy.**—Congenital cases of mental deficiency in which the signs of the morbid state are late in appearing See MENTAL DEFICIENCY (*Developmental*)

**Developmental Insanities.**—Under this name have been grouped the deliriums and night terrors of children and the various insanities of puberty and adolescence See ADOLESCENT INSANITY, NIGHT TERRORS, etc

**Developmental Method.**—The spinal cord at birth has tracts (ongoing) which contain uncollated nerve fibres, while others

(outgoing) do not, by this fact the various tracts of the cord can be demonstrated. See PHYSIOLOGY, NERVOUS SYSTEM (*Spinal Cord, Functions, Conducting Paths*).

**Deviation.**—A departure from the normal state, deflexion or variation, *eg* divergence of one or both optic axes, from the normal position. See OCULAR MUSCLES, AFFECTIONS (*Paralysis, Etiology*), NOSE, DISEASES OF NASAL ORIFICES AND SEPTUM (*Deviation of the Septum Nasi*).

**Devonshire Colic.**—Cider (from its acidity) easily affects lead with which it comes in contact, so lead-poisoning may be produced by drinking cider. This may account for the frequency of plumbism in Devonshire. See TOXICOLOGY (*Instants, Lead*).

**Devoto's Method.**—The use of phosphotungstic acid or tannin for the precipitation of albumoses in urine. See URINE, PATHOLOGICAL CHANGES IN (*Detection of Albumoses*).

**Dew.** See METEOROLOGY (*Dew and How Frost*).

**Dextrocardia.**—Transposition (congenital) of the heart to the right side of the thorax, localised or partial heterotaxy, also written dextrocardia. See HEART, CONGENITAL MALFORMATIONS (*Dextrocardia*).

**Dextrin.**—A carbohydrate ( $C_6H_{10}O_5$ ), called British gum, closely allied to mulin, obtained from starch by the action of dilute acids, of diastase, and of animal ferments, it is a polysaccharide, got by the polymerisation of glucose or dextrose ( $C_6H_{12}O_6$ ), the first dextrins formed by the action of saliva on starch give a brown colour with iodine and are called *cyltho-dextrins*, the next give no colour (*achroodextrins*). See PHYSIOLOGY, FOOD AND DIGESTION (*Food, Carbohydrates, Polysaccharids*).

**Dextrose.**—(Glucose, grape sugar, or blood sugar, the aldehyde of mannite, a simple carbohydrate ( $C_6H_{12}O_6$ ), or monosaccharid, it is so called because it is dextro-rotatory, *ie* rotates the plane of polarised light to the right, and thus differs from levulose, which it otherwise resembles, it is one of the aldehyde sugars or aldoses. See GLYCU-SURIA, PHYSIOLOGY, FOOD AND DIGESTION (*Carbohydrates*).

**Dhobie Itch.** See SKIN DISEASES; OF THE TROPICS (*Vegetable Parasites*).

**Diabetes.** See ADRENAL GLANDS, ADDISON'S DISEASE (*Diagnosis, Browned Diabetes*), ALCOHOL (*Indications, Diabetes*), ALOPECIA (*Etiology, Diabetes*), BOILS AND CARBUNCLE (*Etiology*), BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Cerebral Hemorrhage, Diagnosis from Diabetes*), BRAIN, SURGERY OF (*Compression of the Brain, Diagnosis*

*from Diabetic Coma*), BREATH, BRONCHI, BRONCHITIS (*Etiology, Predisposing Causes*), CATARACT (*Idiopathic, Causes*), CLIMATE, ACCLIMATISATION (*Diabetes*), COLON, DISEASES OF (*Membranous Colitis, Secondary*), COLOUR VISION (*Acquired, Causes*), DIABETES INSIPIDUS, DIABETES MELITUS, EAR, MIDDLE EAR, CHRONIC SUPPURATION (*Causes*), GANGRENE (*Diabetic*), INSANITY, ETIOLOGY OF (*Causes, Autotoxic*) INSANITY, PATHOLOGY OF (*Pathogenesis, Diabetic Insanity*), INSANITY, NATURE AND SYMPTOMS (*Etiological Varieties*), INVALID FEEDING (*Cookery in Diabetes*), LUNGS, GANGRENE OF (*Causes*), LUNGS, VASCULAR DISORDERS (*Pulmonary Embolism, Fat Emboli*), MILK (*Therapeutic Uses*), MORPHINOMANIA AND ALLIED DRUG HABITS (*Paraldehyde Habit, Diagnosis*), MUSCLES, DISEASES OF (*Polymyositis, Etiology*), NAILS, AFFECTIONS OF THE (*In General Diseases*), NERVES, PERIPHERAL (*Neuritis, Causes*), NERVES, MULTIPLE PERIPHERAL NEURITIS (*Etiology*), NERVE, NEURALGIA (*Etiology*), OCULAR MUSCLES, AFFECTIONS OF (*Paralysis, Etiology*), PANCREAS, PHYSIOLOGY OF (*Relation of Diabetes Mellitus to Lesion of Pancreas*), OVARIES, DISEASES OF (*Ovariectomy in Diabetic Subjects*), OXYGEN, USES OF, PENIS, SURGICAL AFFECTIONS OF (*Balanitis*), PHARYNX, CHRONIC PHARYNGITIS (*Etiology*), PHYSIOLOGY, INTERNAL SECRETIONS (*Suprarenal Bodies, Pancreas*), PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Digestive, Diabetes*), PRURITUS (*Etiology*), PUBERTY, PHYSIOLOGY (*Excretory System, Glycosuria*), REFRACTION (*Hypermetropia*), RETINA AND OPTIC NERVE (*Retinal Hemorrhages, Causes, Diabetic Retinitis*), SKIN, PIGMENTARY AFFECTIONS OF (*Classification, Toxicum*), TEETH (*Dental Caries, Causes*), THERAPEUTICS, HEALTH RESORTS (*Warm Climate for Diabetes*), UNCONSCIOUSNESS (*Auto-intoxications*), URINE, PATHOLOGICAL CHANGES IN (*Ammonia, Phosphates, Sugars, and Acetone in Urine*), X-RAYS (*Diabetes*), XANTHOMA (*Xanthoma Diabeticorum*).

## Diabetes Insipidus.

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DIABETES INSIPIDUS is a disease characterised by a prolonged morbid increase in the secretion of a urine free from sugar.

**ETIOLOGY.**—Diabetes insipidus is a comparatively rare condition. During a period of six years twenty cases were under treatment in the wards of the Royal Infirmary, Edinburgh. According to the statistics of Roberts, Strauss, and van der Heyden, the disease is most common during adolescence, early manhood, and middle life. Sex seems to play a not unimportant part as a predisposing factor, males being twice as frequently affected as females. A marked family predisposition may exist, the disease

running through neuropathic families or alternating with other nervous or mental troubles. Thus Griesinger in one instance found that seven blood relations of the patient were epileptic, in two instances the patient himself was epileptic, in one instance both the patient and his friends were epileptic. Gee notes a family where in four generations of 23 patients 11 suffered from diabetes insipidus, Ossi, a family of 9, 6 of whom were affected, and Weil, a family of 21, 20 of whom developed the complaint. The causal relationship between syphilis and diabetes insipidus is not yet quite clear. In most cases the condition must be ascribed to cerebral gumma or endarteritis, but a number remain where the exact condition cannot be substantiated. In brain diseases tuberclelosis must not be forgotten in diabetes insipidus in childhood, and two cases have been described where chronic hydrocephalus existed.

The acute infectious diseases, gout, trauma of the head or abdomen, mental shock and worry, exposure to cold, a bout of drunkenness, neglect and improper feeding, with consequent malnutrition in children, may all have a causal relationship to the disease.

**SYMPTOMS**—Two prominent and characteristic symptoms are present in the disease—an increase in the amount of urine excreted and a marked and distressing thirst. The onset of the disease may be insidious or sudden, there is complaint of increased frequency of micturition and increase in the amount of urine passed, distressing during the day, and preventing proper sleep during the night. The amount of urine is enormously increased, as much as fifteen to twenty pints being passed in twenty-four hours. Except in the later stages of the disease the amount of urine excreted exceeds considerably the amount of fluid ingested, the surplus being made up from the food and the tissues of the body. When, however, a certain stage of the disease is reached the tissues become dehydrated, and this disproportion is lost. The urine is pale in colour with a yellowish or greenish tinge. The reaction is faintly acid or neutral. The specific gravity is always low—1002 to 1005. Glucose is absent, but mosite or muscle sugar is frequently present, but its presence is not characteristic, as it may be found in chronic interstitial nephritis, and even in health after an excessive amount of water has been drunk. As a rule albumin is absent, except in the later stages of the disease, when it may be present as the result of the general dyscrasia and disorganisation of the kidneys. The urine on standing may become turbid from the deposit of epithelial cells, crystals of oxalate of lime may be present, and phosphates may form from ammoniacal decomposition. The percentage of total solids is small, but the total daily excretion of nitrogen may be slightly increased. The chlorides and phosphates are increased. The

thirst is excessive and distressing. Enormous amounts of fluids can be swallowed by the sufferer. Trousseau recounts the case of a young man who daily drank forty litres of fluid and passed forty-three litres of urine. Under treatment, when unable to get water the thirst became so intolerable that he drank the contents of the chamber-pot.

The mouth, tongue, and fauces are dry and parched. The appetite may be unaffected, but it is usually increased, and in the early stages of the disease may be voracious. Digestion in the earlier stages of the disease is normally performed. The bowels may be constipated. The temperature is as a rule subnormal. The skin is dry and harsh. The nervous system is disturbed. When the condition is due to a gross cerebral lesion paralysis of cranial nerves with hemianopsia may be present. The sensory functions are usually abnormal, thus pains in the back and legs and a tendency to headache, tingling and itching of the skin may be present. Amblyopia is common without any discoverable lesion. The mental functions are below par, the patient being irritable and depressed. Insomnia is distressing. The sexual powers are enfeebled.

The progress of the disease is variable. At times the condition is so slight as to be regarded more as an annoyance than as a disease. In more severe cases progressive deterioration in nutrition sets in, the muscles waste, the appetite diminishes, digestion fails, and attacks of diarrhoea supervene. The patient becomes exhausted, drowsy, and gradually comatose, unless carried off by an intercurrent attack of hypostatic pneumonia.

**DIAGNOSIS** is not as a rule difficult. The increased excretion of water in diabetes mellitus, intermittent hydronephrosis, chronic interstitial nephritis, and in hysteria, may occasionally simulate that symptom in diabetes insipidus, but these conditions are otherwise readily differentiated.

The *prognosis* in diabetes insipidus is very variable. Each one must be judged upon its own merits. At times the health does not suffer appreciably. Cure may sometimes follow an intercurrent affection such as measles. Idiopathic cases may last for many years, and terminate in a gradual loss of nutrition or in an intercurrent attack of pneumonia or with a slowly progressing tuberculosis. Once anorexia sets in the end is not far distant. When a gross cerebral lesion is present the diagnosis is bad. When the disease appears in childhood the general development may be retarded and deficient.

The *pathology* of diabetes insipidus consists essentially of an error in the controlling power of the vasomotor nerves of the kidney. In animals polyuria has been shown to follow stimulation of the floor of the fourth ventricle or cerebellum. In man the stimulation may



result from peripheral irritation, an abscess of the external auditory meatus has been known to produce marked polyuria, which subsided on evacuation, to return when the free discharge of pus was obstructed, to disappear again as healing took place. In animals section of the great splanchnic nerve is followed by persistent hypersecretion from the kidney on the same side. In man Schapiro has described degeneration and atrophy of the ganglionic cells of the semi-lunar ganglia, and degeneration and destruction of the axis cylinders of the great splanchnic nerve in diabetes insipidus. Various lesions of the brain may be present, as tumours, tubercle, degeneration and softening following arterial changes. There is no characteristic lesion in the kidneys, though in long-standing cases secondary changes may take place as the result of the prolonged excessive secretion of urine.

**TREATMENT**—The treatment of diabetes insipidus, founded as it is upon an imperfect pathology, is not entirely satisfactory. When a definite dyscrasia exists, such as tuberculosis or syphilis, as a causal factor, treatment must be directed to its amelioration by suitable remedies. Apart from this, treatment by drugs has, for the most part, been directed to influencing the condition through the nervous system. Valerian was advocated by Trousseau in enormous doses of 10 grms per diem, progressively increasing to 30 grms. Given thus it inevitably results in gastric disturbance. Smaller doses of 5ss to ʒj of the tincture three daily, with camphor water or spirits of chloroform to disguise the taste, produce a decided, but unfortunately but passing improvement, and any effort at increased dosage is resented by the stomach. Ergot has a decided anorchiating influence, and may be suitably prescribed as ergotin in 2-grain doses in pill with extract of belladonna, given every six hours, the effects being watched lest ergotism be produced. Nitroglycerine, again, has produced decided benefit in a number of cases. Antipyrin may prove useful at night as a sedative, and temporarily diminishing the excretion of urine, and thus permitting sleep. Salts of iron, arsenic, zinc, strychnia, belladonna, nitrate of silver, carbolic acid, and the bromides have all been prescribed with varying success. Drugs, in fact, may diminish the excessive secretion of urine for a time, and have a beneficial effect on the general health, they do not cure the disease. Ergot, belladonna, and nitroglycerine are most to be recommended. Electricity has had a marvellous curative effect in some instances. It may be applied to the medulla, the positive pole of the galvanic current being applied to the back of the neck by a large electrode, the negative pole, suitably insulated to within a quarter of an inch of its extremity, being passed along the floor of the nostril to reach the spine. The strength of the current should be gradually increased from one to

five milliamperes, the duration of the application from one to five minutes. The negative pole may be applied to the epigastric region instead of to the posterior wall of the nasopharynx.

Diet and general hygienic measures are of great moment in the treatment of the disease. It is unnecessary and cruel to needlessly restrict the amount of fluid drunk, and only leads to deterioration in the general health. The daily consumption of fluid should be ascertained, and a gradual diminution advised till it is found that the urinary excretion is no longer influenced. Thirst should be alleviated with ice, acidulated drinks, and the use of pilocarpine in small doses. Alcohol, aerated waters, potash water, tea, and coffee must be avoided on account of their diuretic acid. Food should be nutritious and easily digested. There is no objection to carbohydrates, nor to proteid food within the limits of the patient's powers of digestion. If restriction be placed upon the amount of food a careful watch must be kept upon the patient's weight. It is better that food should be taken frequently in smaller quantity than in large quantities at longer intervals. Little salt must be taken in the food. In the later stages, when anorexia supervenes, the fluids drunk should be nutritious, as milk, cocoa, whey, light broth.

General hygiene is of importance—gentle exercise, a bracing climate, and, as the temperature is subnormal, warm clothing are all important. In the later stages carefully regulated massage is of great use. Great care should be taken of the skin, which becomes atrophic and unhealthy. Dryness may be relieved by steam or warm water baths. Gentle friction aids nutrition. Intercurrent affections must be treated on general principles. Constipation is frequently troublesome, diastolic and irritant purgatives must be avoided from their liability to set up severe diarrhoea. Collection of faeces occurs in the lower bowel, and is best treated by large enemata, massage to the abdomen, about 1½ grain in pill after meals, or fluid extract of cascara sagrada in half-drachm doses at night.

### Diabetes Mellitus.

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DIABETES MELLITUS is a disease in which grape-sugar is persistently excreted in the urine. The

term cannot be applied, however, to all cases in which sugar is detected in the urine. The cases of temporary glycosuria are to be excluded, so also are cases in which the sugar excreted is not grape-sugar, as, for example, lactosuria and pentosuria. The name diabetes mellitus is applied by many writers to all forms of *permanent* glycosuria. Others use the term chronic glycosuria for the milder forms of the disease, in which grape-sugar is present in the urine, but other symptoms slight or absent, and reserve the term diabetes mellitus for the more severe forms.

**PHYSIOLOGICAL CONSIDERATIONS.**—The carbohydrates derived from the products of digestion are conveyed by the portal vein to the liver. This organ contains a carbohydrate, glycogen, which is greatest in quantity when a carbohydrate diet is given. What the exact function of the liver may be, whether it is constantly paying out a small quantity of sugar into the general circulation, or whether the function of the liver and the intestinal villi is to prevent sugar passing into the general circulation, is a disputed point which will be discussed in the article on the liver functions.

Pavy thinks that if sugar were continually being passed into the general circulation, the urine would always contain sugar in quantity. He believes that any excess of sugar in the blood is always eliminated in the urine.

It has been shown, however, that the sugar in the blood disappears when the liver is excised (Minkowski), or when the vessels of the liver are ligatured (Boek and Hoffmann, Segen).

Intravenous injection of large quantities of sugar have not been followed by glycosuria (Biedl and Kraus). Also subcutaneous injections of certain kinds of sugar—dextrose, levulose, and galactose—were not followed by glycosuria, but after the subcutaneous injection of cane and milk sugar, the whole of these substances was found again in the urine ( Voit ).

**EXPERIMENTAL DIABETES AND GLYCOSURIA.**—In this article it is only possible to very briefly mention some of the more important results of experiments on animals.

C. Bernard produced diabetes by puncturing the floor of the fourth ventricle. It appears probable that changes are thereby produced in hepatic cells, and that the impulses which cause them pass along the splanchnic nerves. By other experimental lesions of the nervous system diabetes may be produced, as, for example, by division of the medulla (Pavy), injury of the vermiciform process of the cerebellum (Eckhard), injury of various parts of the pons and posterior columns of the spinal cord (Schiff), centrifugal vagus irritation (Arthaud and Butte). Pavy produced diabetes by injection of dehydrated arterial (oxygenated) blood into the portal vein. Diabetes can also be produced both in man and animals by the administration of phloridzin (v. Mering).

Alimentary, puerperal, and symptomatic gly-

cosuria, as well as glycosuria produced by phloridzin and chemical substances, will be considered in the article on glycosuria.

**PANCREATIC DIABETES.**—It was not until 1889 that it was found by Minkowski and v. Mering in Germany, and by De Dominicis in Italy, that total extirpation of the pancreas in dogs is followed by diabetes. Partial extirpation, i.e. when one-quarter or one-fifth of the gland is left behind, does not produce diabetes. Even when the pancreatic duct is ligatured and the remaining piece of the pancreas has no connection with the duodenum, still diabetes does not follow. Minkowski has shown the relation of the pancreas to diabetes by the most striking experiment of transplanting a piece of the gland and grafting it under the skin of the abdominal wall (external to the abdominal cavity). If the transplanted portion of pancreas (or graft) does not necrose, then diabetes will not occur when the whole of the remaining intra-abdominal part of the gland is removed. But if the transplanted portion of pancreas be subsequently removed, then diabetes occurs.

It is probable that something is formed in the pancreas which passes into the circulation and brings about sugar destruction, or prevents the accumulation of sugar in the blood. Lapine and others believe that this "something" is an internal secretion of the pancreas, which is absorbed by the pancreatic lymphatics and veins.

[It is well known that sugar is broken up in the muscles, presumably by ferment-action, yet no glycolytic ferment can be obtained from them. Cohnheim, however, has shown that if to muscle juice the fluid expressed from the pancreas be added, the mixture has a marked glycolytic power which neither of its components possesses separately. It thus appears as though the internal secretion of the pancreas influences the metabolism of sugar indirectly, through action on the muscles.]

**ETIOLOGY AND EPIDEMIOLOGICAL RELATIONS.**—The disease is more common in males than females, the liability of the two sexes is about equal in the early period of life, but after the age of 30, males are more frequently affected than females. The disease is more common in adults. In private practice it is most frequently met with between the ages of 50 and 60, but in hospital practice there is a larger proportion of young diabetic patients.

The following table gives the age and sex of 100 cases of diabetes in Manchester (mostly hospital patients) —

	AGE IN YEARS						
	10-20	20-30	30-40	40-50	50-60	60-70	Total
Males	6	12	14	18	9	8	67
Females	6	18	8	8	3	5	58
	12	25	22	21	12	8	100

The percentage at various ages recorded by several authors is as follows —

	Under 10	10-20	20-30	30-40	40-50	50-60	60-70	70-80
Diab.		1.7	2.8	11.2	21.1	27.5	15.1	3.1
Sengen	0.5	3	16	16	24	30	10	0.5
Fretsch	1	7	10	15	25	30	11	1

The disease is comparatively rare. At the Manchester Royal Infirmary during the twenty years 1875-93 the number of medical in-patients was 27,721, and of these only 272 suffered from diabetes, *i.e.* only 0.9 per cent.

In India, Ceylon, South Italy, and Malta, the disease is much more common than in most other countries. In India it is more common among the Hindus than the Mohammedans. It is said to be more common amongst the Jews, but probably it is the wealthy Jews who chiefly suffer.

The reports of the Registrar-General show that in England the mortality from the disease is steadily increasing. A similar increase in the mortality has been noted in France, Denmark, and the United States.

**PREDISPOSING AND EXCITING CAUSES.**—Sometimes there is a *family history* of diabetes (13 per cent). Brothers or sisters occasionally suffer, sometimes an uncle or aunt of the diabetic patient has been similarly affected, but it is very rare to find that the father or mother has had the disease.

After *external injuries* temporary glycosuria sometimes occurs, and occasionally a true diabetes follows. It is evident that there must be some other factor in the causation. Still sufficient cases are now on record to show that probably an injury is sometimes the exciting cause (6 per cent of diabetic patients). In at least half of the cases of traumatic diabetes the head has been the seat of the injury.

Numerous striking instances are on record in which diabetes has rapidly followed *fright*, *violent passion*, or *mental emotion*, also in many cases the symptoms have followed prolonged mental anxiety and worry, owing to loss of money and loss of employment, etc. (this history obtained in 10 per cent of cases). Mental anxiety and over-work associated with the nursing of a sick relative, etc., is sometimes an exciting cause (8 per cent of cases).

*Obesity* is sometimes associated with a mild form of diabetes in individuals between the ages of 40 and 60. In young persons occasionally great obesity is followed by a severe form of the disease. (Great obesity preceded diabetes in 4 per cent of writer's cases.)

*Gout* may be associated with diabetes which is usually of a mild form. Such cases are met with chiefly in private practice and are rare amongst hospital patients.

A marked history of *alcoholism* (chiefly beer-drinking) is sometimes obtained (17 per cent of

cases), and probably this is an occasional cause of the disease.

An attack of influenza, an acute febrile affection, pneumonia, bronchitis, pleurisy, exposure to wet and cold, the drinking of cold fluid when the body has been very hot, and injury from a lightning stroke, have been regarded as occasional exciting causes of the disease. (Diabetes developed directly after an attack of influenza in 8 per cent of cases collected by writer.) It is possible that in a few cases *syphilis* may be an indirect cause of diabetes by producing cerebral or pancreatic lesions.

Occasionally diabetes follows *pregnancy* or the development of an abscess of the breast (7 per cent of writer's cases). Occasionally diabetes develops during pregnancy, but ceases with the termination of the latter, to recur at a later date. It has been thought by some writers that the climacteric period favours the occurrence of diabetes in women. In at least 15 per cent of the cases *no history of any exciting cause* can be obtained.

**RELATION BETWEEN DIABETES MELLITUS AND DISEASES OF THE LIVER.** From the results of physiological experiments, one would expect that there would be some clear relationship between diabetes and pathological changes in the liver, but no definite or constant pathological change is met with in the liver in diabetes, though this organ is sometimes diseased. In 20 cases in Manchester, the liver, as regards size, was enlarged in 11, diminished in 4, and normal in 5. In one of the cases multiple abscesses were present, in another there was cirrhosis, in another fatty infiltration and emphysema, in another fatty infiltration and congestion. In the other cases, beyond variations in size, the only change was congestion, which was often present. These are the most common abnormalities, they are not constant, and are very often met with unassociated with diabetes.

The glycogen in the liver cells removed by a fine trocar during life was in one case considerable, but in another case it was absent (Ehrlich). At present *pathological anatomy* does not furnish any evidence that diabetes is related to hepatic changes.

**RELATION BETWEEN DIABETES MELLITUS AND AFFECTIONS OF THE NERVOUS SYSTEM.**—It has been already mentioned that diabetes has often followed great mental anxiety, worry, or sudden fright, and there are many points in favour of the connection of diabetes with some change in the nervous system. But important pathological lesions have been met with in only a small proportion of cases.

The changes usually recorded are slight, and such as are frequently met with when no symptoms of diabetes have been present during life.

A number of cases are on record, however, in

which cerebral changes have been found post-mortem, that have probably been the cause of the diabetic symptoms. In 30 of such cases recorded in literature, there was a tumour in the floor of the fourth ventricle in 4, other changes at this region (such as softening, fatty degeneration, sclerosis, haemorrhage, cysticercus) in 14, tumour of the medulla in 3, lesion at the base of the brain (tumour, softening) in 3, tumour of the pituitary body 1, cysticercus in cerebellum 1, softening in cerebellum 1, cerebral tumour (temporo-sphenoidal lobe) 1, tumour compressing right vagus nerve 2

In 14 consecutive cases recently examined by the writer, the medulla and other parts of the brain appeared normal in 9, in 5 naked-eye changes were found. In the latter cases the changes were the following: cyst of the cerebellum close to the right vagus root in 1, cerebro-spinal meningitis (probably secondary to hepatic abscess) in 1, minute hemorrhagic patch in left vagus nucleus in 1, tumour of the pituitary body in 2 (in those two cases symptoms of acromegaly in addition to those of diabetes were present during life)

Careful microscopical examination of the medulla has been made frequently, but usually with negative results. The minute excavations around the cerebral arteries described by Dickinson are now generally believed to be due to the effects of hardening. French described a marked dilatation of the small vessels of the medulla, and this he regarded as the most important and constant change in the nervous system in diabetes. Of 10 cases of diabetes examined microscopically by the writer, the medulla appeared normal in 4, in 1 there was purulent meningitis, in 5 cases the blood-vessels of the vagus nuclei were much dilated. In 3 of the latter 5 cases there were small hemorrhages present in the vagus nuclei, twice unilateral, once bilateral. In one of the cases the hemorrhagic patch could be seen with the naked eye.

Though the examination of the medulla often yields negative results, it is still possible that minute or functional changes may be present in the nerve cells of the vagus nuclei, or at other part of the medulla, which cannot be recognised at present by microscopical examination.

The spinal cord is usually normal, or presents changes which are to be regarded as secondary, but in a very few cases gross lesions have been found which may have been the primary cause of the symptoms (tumour or softening of the cervical region).

The sympathetic nerves and ganglia (cervical and abdominal) have been carefully examined by Hale White and others, often they have been normal, and when changes have been found they have not differed from those which have been frequently met with when there have been no diabetic symptoms during life.

Occasionally a mild glycosuria occurs in association with well-marked disease of the nervous system, such as locomotor ataxia, disseminated sclerosis, Graves' disease, etc. But the association of a well-marked diabetes with these affections is very rare.

**DIABETES AND ACROMEGALY**—During the last five years a number of cases of acromegaly have been recorded in which glycosuria was present, and in some of the cases there has been a well-marked diabetes (2 cases of diabetes associated with acromegaly have come under the writer's observation). Out of 21 cases of acromegaly recorded in literature, 4 were associated with true diabetes, 2 with glycosuria, and in 15 cases the urine was free from sugar. In acromegaly a tumour of the pituitary body is usually present, and cases of tumours of the pituitary body are on record in which diabetes has been present, but symptoms of acromegaly absent.

**THE RELATION OF DIABETES TO LESIONS OF THE PANCREAS**—Changes in the pancreas have been described from time to time, ever since Cawley recorded a case of diabetes (in 1788) in which the pancreas was atrophied and contained calculi. Many years ago Lancereaux drew attention to the pancreatic lesions.

In 24 consecutive cases of diabetes examined pathologically by the writer, the condition of the pancreas was as follows—

	Cases
(a) Extensive changes (very marked cirrhosis 2, cancer 1, extensive atrophy 1)	4
(b) Well-marked changes (cirrhosis 2, lipomatosis 1, atrophy with fatty degeneration and infiltration 1)	4
(c) Slight changes (atrophy with slight fatty degeneration 1, atrophy out of proportion to the general wasting 2)	3
(d) Atrophy, but only in proportion to the general wasting	5
(e) Pancreas normal, macroscopically and microscopically	8
	<hr/> 24

In the first 4 cases of the above table the pancreatic changes were so extensive that, bearing in mind the result of total extirpation of the gland in animals, there can be little doubt that the diabetes was due to the pancreatic lesion.

In the second group of cases it also appears probable that diabetes was due to the pancreatic changes, in the third group the relation is uncertain, and in the last two groups of cases either the diabetes was not dependent on the pancreatic disease, or the pancreatic affection, if present, was a functional one which could not be recognised macroscopically or microscopically.

Many cases of diabetes associated with pancreatic disease are now on record, and various extensive changes have been found, such as cirrhosis and fibroid changes, fatty infiltration

and degeneration, calculi, cancer, cysts, and marked atrophy. Often the pancreatic tissue has been almost absent owing to the changes mentioned.

It is very improbable that all these varied pancreatic changes should be the result of diabetes, and when we consider the remarkable results of experimental removal of the pancreas, it appears very probable that, in certain cases, diabetes is directly due to pancreatic disease.

There are two objections to the pancreatic theory of diabetes in man. The first is that *glycosuria is absent in many cases of diseases of the pancreas*. But it is important to remember that in extirpation of the pancreas in animals, if a small portion of the gland should be left behind, no diabetes follows. And in man, in disease of the pancreas, often only a portion of the gland is affected.

The second great objection to the pancreatic origin of diabetes is the fact that the *pancreas is not affected in all cases of diabetes*. In one-third of the cases the pancreas is normal macroscopically and microscopically. Hence in these cases either the lesion causing the diabetes is not in the pancreas, or the pancreatic affection is a functional one.

It is conceivable that arterio-sclerosis may cause diabetes by producing changes in the pancreas or in the nervous system, and several instructive cases of diabetes are on record in which marked pancreatic changes were found post-mortem that were apparently the result of arterio-sclerosis.

[The conflicting statements which have been made concerning the relation of pancreatic disease to diabetes have now been to a large extent reconciled by the work of Opie and others. There is good reason to believe that one pancreatic lesion, and one only, has the power of so influencing the action of the internal secretion of the organ on carbohydrate metabolism that glycosuria results, and that is disease of the ductless islands of Langerhans. These bodies are involved in interlobular pancreatitis, not in the interlobular form, and only when their cells are invaded, whether by this process, be it primary or secondary to arterio-sclerosis, hepatic cirrhosis, or cancer, or by some lesion involving the whole pancreas, does diabetes result.]

**DIABETES OF ENDOGENOUS ORIGIN**—In many cases of diabetes the most careful inquiry fails to reveal any exciting cause, and often the most careful examination of the brain, pancreas, liver, and other organs fails to reveal any changes except what are secondary or accidental.

It is quite possible (as suggested by Strumpell) that some cases are entirely or almost entirely endogenous in origin, i.e. they are due to some developmental abnormality.

#### SYMPTOMATOLOGY

In the severe forms of diabetes the face is

often wasted in appearance, the wrinkles and naso-labial folds are well marked, and the expression is often anxious or sad. But the face and lips are not anæmic, or not markedly anæmic, unless some complication should be present. In the mild forms of the disease, however, the facial expression is not characteristic.

In severe forms of diabetes *wasting* is a prominent symptom, especially in young persons, whilst in elderly patients mild diabetes is often associated with *obesity*.

**ONSET**—In many cases *thirst* and *diuresis* are the first symptoms noticed, sometimes the earliest sign is troublesome *cramps* in the calf muscles at night. In other cases the patient first seeks medical advice on account of *wasting* and *increasing weakness*, or on account of one of the complications (gangrene, carbuncle, cataract, eczema of the genital organs, etc.). Occasionally the patient states very definitely that the thirst commenced suddenly on a certain day, and at a certain hour. Sometimes a very slight glycosuria, unaccompanied by thirst, diuresis, or other symptoms, has preceded the true diabetes for months or years, but certainly this is not always the case.

**THE URINE**—The *quantity* of urine is increased (often 150 to 300 ounces in the 24 hours), but in many of the milder cases the increase may only be slight (60-100 ounces daily).

The amount of urine is about equal to the fluid taken. It is reduced by a nitrogenous diet and by intercurrent disease, and often diminishes before a fatal termination. The *colour* of the urine is very pale, generally light yellow or straw coloured, often, but not always, it has a *paleish-yellow* tint. But when the amount of urine is not increased, in the mild forms, the colour may be normal.

A mucous cloud, when present in diabetic urine, is often seen, not at the bottom, but at the upper part of the urine glass. Diabetic urine is usually bright and clear, but sometimes in female patients it is turbid from the presence of pus and epithelial cells. The *smell* is often sweet or aromatic, and in severe cases, especially just before the onset of coma, the urine has a peculiar "chloroform" smell, usually attributed to the presence of acetone. The *taste* of diabetic urine is sweet. The *reaction* is nearly always acid, and often markedly acid. The *specific gravity* is increased (up to 1030, 1045, or higher). A small quantity of sugar may be found, however, in urine of a normal or low sp. gr.

The *presence of sugar* is of course the most important change in the urine in diabetes. Sometimes before the patient has been aware of the nature of the disease he has been struck by the fact that flies have been attracted to his urine, also, if a drop of it has fallen on to his boot, or any adjacent object, and has been allowed to dry, he has observed that a salt-like

deposit has been left behind. The sugar present in diabetic urine is grape-sugar (glucose), and its amount varies according to the nature of the case from 0.5 to 6, 8, or 12 per cent. The daily amount excreted may be 3000 to 4000 grams, or more. (For sugar tests see articles on "Glycosuria" and "Urine, Pathological Changes.") The sugar increases in mild cases after food, and diminishes during fasting, and hence the excretion is less during the night. In very mild cases sugar may be absent in the night urine (passed before breakfast), whilst it is abundant in the day urine. The sugar excretion is increased by starchy and saccharine foods, and diminished by nitrogenous diet. In mild cases, when carbohydrates are withdrawn from the diet, the sugar disappears from the urine, but returns when carbohydrates are taken again. In the mildest cases simple restriction of the carbohydrates is sufficient to cause the glycosuria to disappear. These are points of practical importance in urine testing in mild cases. In severe cases of diabetes sugar is present in the urine in spite of the withdrawal of all carbohydrate food, and may be present during fasting.

Sugar is the carbohydrate in the diet which causes glucose to be eliminated in the greatest quantity in the urine, whilst starch and other carbohydrates are less injurious. Grape-sugar is most injurious, fruit-sugar (levulose) is only about half as injurious as grape-sugar, milk-sugar and cane-sugar stand midway between grape-sugar and levulose. Fats never increase the sugar excretion, and alcohol in moderate quantity has no effect.

*Muscular exercise* diminishes the sugar excretion in well-nourished patients suffering from a mild form or early stage of the disease. But when the affection is severe, and the patient wasted, exercise increases the sugar excretion.

The sugar excretion is often *diminished or arrested* by intercurrent affections.

The excretion of *urea* is increased owing to the excess of nitrogenous food taken by the diabetic patient, only in a few cases is the increased urea excretion due to the destruction of the albumen of the body (Seegen).

Not infrequently there is a small deposit of uric acid crystals at the bottom of the urine glass in mild cases of diabetes.

The excretion of *ammonia* is increased in some cases, but not in all. In diabetes coma it is greatly increased. Sometimes there is an abundant deposit of oxalate of lime.

At the early stage of the disease *albuminuria* is usually absent, but it frequently appears at a late stage of the disease. Thus in 100 cases the writer found albumen present in 30 when the patients first came under observation, but in some of the cases in which albuminuria was absent at first it developed later, so that finally it was present in 44 per cent. The albuminuria is usually very slight, and not associated with

nephritis or any gross lesion of the kidney, but in a few cases the albuminuria is abundant, and there are signs of parenchymatous or interstitial nephritis. In only 4 out of the 100 cases of diabetes just mentioned was the amount of albumen large. In diabetic coma albumen and casts are nearly always present in the urine, but otherwise the albuminuria is not accompanied by casts, except in the rare case when there are indications of actual nephritis.

When inflammation of the prepuce or of the vulva is present as a complication, the urine, when recently passed, often contains fungus spores and mycelia along with a few pus cells. In diabetic females the urine is not infrequently slightly turbid, owing to the presence of pus and epithelial cells.

In the severe forms of diabetes, when a solution of perchloride of iron is added to the urine, there is at first a turbidity owing to the precipitate of phosphates, but on adding more of the perchloride solution a dark brownish-red coloration is often obtained. This coloration is known as *Gerhardt's reaction*, and is usually attributed to diacetic acid, by some it is attributed not to this substance, but to other closely-allied compounds. This reaction is an indication of a severe form of the disease, it is usually present in diabetic coma, but in mild forms of the disease it is absent. A similar reaction is occasionally met with in febrile diseases and several other affections. Also the urine of patients who are taking salol, salicylic acid, and salicylate of soda gives a dark, brownish-red coloration with perchloride of iron, but the colour has more of a violet or purple tint than that obtained in severe cases of diabetes. The urine of patients taking antipyrin, salipyrin, and several other drugs, also gives a brownish-red coloration with perchloride of iron. When the urine of a diabetic patient gives a reaction with perchloride of iron, often acetone is also present. It may be detected by Legal's test. To several cc of urine a few drops of a concentrated, freshly made solution of nitro-prusside of sodium are added. The mixture is made alkaline with liquor potassæ, and then acetic acid is added when a violet-red coloration is obtained. Another method is to distil about half a litre of urine, to the distillate a few drops of a solution of iodine in iodide of potassium, and a few drops of caustic potash, are added. When acetone is present a precipitate of iodoform occurs. It is yellowish in colour, and has the characteristic iodoform smell, under the microscope it presents hexagonal plates or stars. It has been shown that acetoneuria may be produced in healthy persons by a diet of nitrogenous and fatty food, free from carbohydrates, but on the addition of carbohydrates the acetoneuria disappears (Hirschfeld, Rosenfeld, and others). A high degree of acetoneuria or an increase of the acetone excretion is regarded by Hirschfeld as an indication of approaching coma.

In the severe forms of diabetes  $\beta$ -oxybutyric acid is often found in the urine

**THE BLOOD**—The most important change in the blood is the excess of sugar present. A minute quantity of sugar is present in normal blood (0.6 to 1.0, or a little more than 1.0 per 1000, Favy, average in ten healthy men, 1.7 per 1000, Seegen). In diabetes mellitus the amount of sugar in the blood is usually greatly increased, and may reach 2.7 to 5.7 per 1000.

The writer has discovered a simple method of distinguishing diabetic blood from non-diabetic blood by its reaction with a solution of *methylene blue*. This method is extremely sensitive, and it is only necessary to examine a *drop* of blood obtained by pricking the finger. On heating a drop of diabetic blood and an alkaline solution of methylene blue in *certain* proportions the blue colour of the solution is removed, whilst if non-diabetic blood be used in place of diabetic blood (in the *same proportion*) the solution retains its blue colour.

The test may be performed as follows—A small, *narrow* test-tube is well cleaned, and a drop of water (40 *cubic millimetres*) placed at the bottom. It is important to use a narrow test-tube so that the upper surface of the fluid with which the air comes in contact may be as small as possible. To measure the quantity of blood the capillary tube of a Gowen's hemoglobinometer which is graduated for 20 *cubic millimetres* may be used. The tip of one of the patient's fingers is cleaned and dried, and then pricked. When a large drop of blood has escaped it is sucked up into the capillary tube. Twenty *cubic millimetres* of blood are taken from the finger. The blood is blown gently into the water at the bottom of the small test-tube. Then 1 *cubic centimetre* (*i.e.* 1000 *cubic millimetres*) of a watery solution of methylene blue is added. The strength of the methylene blue solution is 1 in 6000. (To measure the methylene blue solution the 1 cc pipette tube of Southall's meter may be used.) To the mixture of blood and methylene blue in the test-tube finally 10 *cubic millimetres* of liquor potassæ (B.P.) are added. The fluids in the tube are then well mixed by shaking. As a control experiment a second test-tube of similar size is taken, and into this is placed the same quantity of non-diabetic blood with the same proportion of water, methylene blue, and liquor potassæ.

The fluid in each tube has a fairly deep-blue colour. Both small tubes are then placed in a beaker, capsule, or very wide test-tube containing water. Heat is applied by a spirit lamp until the water boils. It is allowed to continue boiling for about four minutes. By the end of this time the fluid in the tube containing the diabetic blood changes its colour from a fairly deep-blue to a dirty pale yellow (almost the colour of normal urine), whilst the fluid in the

tube containing the non-diabetic blood remains blue. Occasionally it becomes bluish-green, sometimes violet, but it never loses its blue colour. The tubes should be kept quite still whilst in the water-bath, as by shaking the decolorised methylene blue is apt to be oxidised by the oxygen of the atmosphere, and a blue tint may then return to the fluid. This is the reason why it is necessary to employ a water-bath, since if the test-tubes be heated directly over a spirit lamp it is difficult to avoid shaking the fluid.

A milky appearance of diabetic blood has occasionally been observed on post-mortem examination. In other cases the blood has had a pink colour, and on standing a milky or cream-like serum has separated on the surface. This cream-like condition of the serum has been shown by microscopical and chemical examination to be due to the presence of fat globules. Analysis of the blood has sometimes shown that the percentage of fat has been increased.

**SYMPTOMS, COMPLICATIONS, AND PATHOLOGICAL CHANGES IN CONNECTION WITH THE VARIOUS SYSTEMS**—Besides the changes in the urine other prominent symptoms are—thirst, increased appetite, great weakness, and emaciation in the severe forms of the disease, a harsh dry skin, often a red, raw-looking tongue. The temperature is normal or subnormal except when complications occur.

**ALIMENTARY CANAL, LIVER, AND PANCREAS**—The saliva is usually scanty and the mouth dry. The gums are often inflamed, spongy, and swollen. The teeth are often carious, and become loose and fall out. Thirst is one of the most characteristic symptoms of the disease, and often enormous quantities of fluid are taken, but in very mild cases the thirst is slight or absent, and also in severe cases during the last few days of life the thirst often diminishes or ceases, when phthisis or other complications have developed. The appetite is generally increased greatly, but this is not such a constant symptom as thirst. In certain mild cases the appetite is not increased, also in severe cases at a late stage the appetite fails.

Diabetic patients very frequently suffer from constipation. In chronic cases diarrhoea is occasionally present.

Pathological examination does not reveal any characteristic change in the stomach or intestines. Occasionally tuberculous ulceration of the intestines has been met with. The pathological changes in the liver and pancreas have been already referred to.

**THE LUNGS**—The most frequent pulmonary complication is tuberculous phthisis. It occurs more frequently in young diabetics, and in the severe forms of the disease amongst the poor. In elderly diabetics, especially in the obese or gouty, tuberculosis is much less frequent.

In 100 consecutive cases (chiefly hospital

patients) in Manchester there were evidences of phthisis in 29.

Next to coma, tuberculous phthisis is the most common termination of diabetes. In half the cases of diabetes in which the writer has seen or made an autopsy, tuberculous disease of the lungs (extensive or slight) has been present. The phthisis of diabetic patients is usually tuberculous, and tubercle bacilli are present in the sputum.

Phthisis in diabetic subjects generally runs a comparatively latent course, and the pathological changes are more advanced than is suspected from the clinical signs. Cough and expectoration are often slight. The temperature is not much above normal, and hemoptysis is very rare. But to these general statements there are exceptions. When the lung changes become advanced the diabetic symptoms gradually subside, and finally the sugar may disappear from the urine.

Pathologically diabetic phthisis is usually due to a chronic caseous tuberculous bronchopneumonia, it generally runs a rapid course, caseation soon occurs, and the diseased parts break down and cavities are formed. There is no tendency to cicatrization.

Occasionally a *chronic pneumonic* (non-tuberculous) phthisis is met with in diabetic subjects.

Gangrene of the lungs occasionally occurs as a complication, in such cases the characteristic offensive smell of the sputum may be absent.

Broncho-pneumonia and acute croupous pneumonia are rare complications.

**THE HEART**—Usually there are no signs of cardiac disease, especially in the severe forms of diabetes. (Heart normal on clinical examination in 93 out of 100 cases examined by the writer.) In the later stages of the disease the heart's action and the heart sounds are often feeble.

Mayer of Carlsbad has found cardiac enlargement in 21.6 per cent of the cases (but probably a considerable proportion of his patients would be elderly persons suffering from a mild form of the disease).

Occasionally diabetes terminates with symptoms of cardiac failure or collapse (see "Diabetic Coma," p. 329).

The pulse is usually normal, but in elderly persons often signs of arterio-sclerosis and high tension are met with, and sometimes the pulse is large, hard, and of increased tension in diabetics under middle life, even when there are no evidences of kidney disease.

Pathologically in severe forms of the disease the heart usually presents no macroscopical change except general atrophy, or the heart muscle is often soft and flabby. Israel, however, found cardiac hypertrophy (generally associated with hypertrophy of the kidneys) in 10 per cent of the cases of diabetes examined pathologically at the Berlin Pathological Institute.

Sometimes fatty or glycogenic degeneration of the cardiac muscle is found on microscopical examination.

**THE KIDNEYS**—Albumen, usually in small quantities or mere traces, is often present in the urine of diabetic patients (44 per cent of cases). It occurs especially at the late stages of the disease, and is not associated with nephritis. It has been attributed to excess of nitrogenous food, to catarrh of the bladder, and occasionally it is due to the mixture of a little pus with the urine owing to the balanitis in the male, or eczema of the vulva in the female. In a few cases the albumen is large in amount, and is associated with signs of nephritis, parenchymatous or interstitial (4 out of the 44 cases of albuminuria just mentioned).

On pathological examination hypertrophy of the kidneys is not uncommonly found. Occasionally there are signs of interstitial, parenchymatous, or diffuse (parenchymatous with interstitial) nephritis, and in a few cases tubercle of the kidneys. Microscopically changes in the renal epithelium are often present, and the following have been described—hyaline degeneration (Altmann), necrosis of epithelium (Ebstein), fatty degeneration (Fichtner); glycogenic degeneration (Ehrlich and Fierichs). The latter change is met with chiefly in the cells of Henle's loop.

**THE SKIN**—In severe cases the skin is usually dry and rough, but in the milder cases it is generally normal. Pruritus is sometimes troublesome, it is usually local, rarely general. Pruritus is most common about the genital organs, especially in females, and sometimes it is the symptoms for which advice is first sought. Pruritus of the vulva is produced by the irritation of the saccharine urine, it is often followed by erythema and eczema. In the male pruritus of the glans penis is due to the same cause, and is often followed by erythema and balanitis, and the prepuce may become oedematous.

Boils and carbuncles sometimes occur, the latter are most frequently met with on the neck. In patients over 45 gangrene sometimes occurs. It may be moist or dry, and is frequently associated with arterio-sclerosis. It occurs most frequently in the foot, and is often excited by some slight wound or injury to the skin. Gangrene may follow cellulitis in other parts.

Perforating ulcers on the feet resembling those seen in locomotor ataxia are occasionally met with in diabetics over middle age.

Anasarca of the feet and sometimes of the hands, without albuminuria or signs of cardiac failure, is an occasional complication of diabetes.

Xanthoma diabeticorum is one of the rarest skin affections in diabetes.

Other skin affections are occasionally met with, and recently a form of diabetes with bronzing of the skin has been described (*diabète bronzé*).

**AFFECTIONS OF THE EYE**—Defects of vision



are not uncommon in diabetes. The most important affection is cataract (9 per cent of cases). It is usually bilateral, and occurs in young patients as well as in the aged. It is generally of the soft variety, but not invariably.

Defects of vision are also caused by paresis of accommodation, shortsightedness developing late in life, vitreous opacities, retinitis, and amblyopia. Diabetic retinitis is very rare (seven cases in 100). It only occurs in patients over the age of 40. In one form of diabetic retinitis the changes consist of small punctiform hemorrhages scattered over the retina. In another form the changes consist chiefly of small white patches chiefly near the centre of the retina. These white patches have a "curdy" appearance, and are sometimes clustered together in the form of a semicircle or incomplete circle surrounding the yellow spot, they are never grouped in a star-shaped form radiating from the yellow spot as in albuminuric retinitis.

Occasionally diabetic patients suffer from amblyopia with defect of vision chiefly in the centre of the field and with central scotomata for colours. Ophthalmoscopic examination reveals no changes in the disc or retina, and the condition resembles tobacco amblyopia.

**SEXUAL FUNCTIONS**—In the male, diminution or loss of sexual power is not infrequent. In females, the sexual desire is said to be diminished greatly in severe cases, whilst in the mild forms of the disease in elderly women it is said to be increased. Amenorrhœa sometimes occurs at an early stage. Pregnant diabetic women often abort, and during pregnancy and the puerperal state the disease often advances markedly.

**NERVOUS SYSTEM**—Mental dulness and drowsiness are frequent. Often the patient is melancholic and depressed, but as a rule the intellect remains clear up to the last. The writer has recorded two cases in which changes were present in the posterior columns of the cord in diabetic patients. In both cases the changes were seen clearly with the naked eye. After hardening in Muller's fluid the degenerated tracts were paler in colour than the rest of the white matter, in both cases they occupied Goll's columns in the cervical region, they extended into Burdach's columns in the dorsal region in one case, in the lumbar region in the other. Microscopically the changes were much less distinct, and consisted in slight degeneration of the fibres of the posterior columns. Probably they were the result of the diabetic blood condition. Similar changes have been recorded by several observers. *Cramps* in the calves of the legs at night are common in diabetes, and are sometimes amongst the earliest symptoms of the disease. The patient may also complain of gnawing pains in the legs, which may be so tender that he cannot bear one leg to lie over the other in bed. The legs may feel numb, and the knee-jerks in such cases are often absent. These symptoms are probably

due to slight peripheral neuritis. Occasionally, but very rarely, there are well-marked symptoms of peripheral neuritis—the feet being "dropped" and the legs paralysed, and sensory symptoms being present as in ordinary alcoholic neuritis. Cases are on record of monoplegia and of paralysis of single groups of muscles which are probably of neurotic origin.

A small number of cases have been published in which pathological examination has verified the diagnosis of peripheral neuritis (parenchymatous form). In most of the cases recorded the neuritis has occurred in patients over the age of 50, and this complication does not bear any relation to the amount of sugar in the urine.

The *knee-jerks* are not infrequently lost in diabetic patients, and in such cases there are sometimes slight signs of neuritis (gnawing pains in the legs, tenderness, and cramps in the calf muscles), very rarely there are marked signs of *peripheral neuritis*. But frequently when the knee-jerks are lost there are no other indications of neuritis, and pathological examination may reveal no changes in the peripheral nerves. Amongst 100 diabetic hospital patients the writer found the knee-jerks both lost in 49, both present in 45, one present, one absent in 6. Most of these patients suffered from a very severe form of the disease, with great wasting. In private practice, where most of the mild forms and early cases are met with, the knee-jerks are only lost in a small proportion of cases. The knee-jerks when present at an early period are frequently lost later. During the last few days of life the writer found the knee-jerks lost in 73 per cent of hospital cases, and they were lost in 20 out of 23 cases of diabetic coma. In the severe forms of the disease, when the knee-jerks have been absent, the writer has usually found the wrist-jerks also absent. But the superficial reflexes—plantar, abdominal, and epigastric—are generally readily obtained or increased, even when the knee and wrist jerks are absent.

**DIABETIC COMA**—The most frequent termination of diabetes is in coma, which is not dependent on any gross lesion of the organs. When advanced phthisis is present as a complication, usually coma does not occur, but to this rule there are exceptions.

Diabetic coma may occur in all forms of the disease and at all ages, but it is especially common in young patients and in the severe forms. Coma may develop at a very early date after the onset of diabetes, especially in young people (few weeks or months), or it may only occur after the disease has been present for years. Coma has frequently developed directly after a long railway journey. Great muscular exertion, great mental worry and anxiety are also exciting causes. A sudden change of diet and a very rigid diet are thought by many (Elstern, Naunyn, Schmitz, Grube, etc.) to

favour the development of diabetic coma when the patient is suffering from a severe form of the disease, and when the urine gives a marked reaction with perchloride of iron. Prolonged constipation is probably a predisposing cause, and numerous cases are on record in which various complications and also surgical operations have appeared to be exciting causes.

*The symptoms of diabetic coma* often commence with lassitude, epigastric pain, nausea, and occasional vomiting. In other cases shortness of breath is the earliest symptom, and it has occasionally preceded other symptoms for several days. Headache may occur at the onset. Often the patient is anxious, restless, or excited at first, then drowsiness gradually develops and passes into coma. The pulse becomes rapid and feeble, and Lépine regards rapidity of the pulse as an important early sign of commencing coma. Dyspnoea is a prominent feature in the majority of cases, and the breathing has a peculiar panting or sighing character, the number of respirations per minute is often only slightly increased or even normal, and the dyspnoea is often indicated by deep inspiration and deep expiration, rather than by much increase in the frequency of respiration. This peculiar dyspnoea is described by Kussmaul as air-hunger. The bowels are generally constipated. The skin becomes pale and cold, and in many cases there is slight cyanosis of the nose, lips, ears, hands, and feet. The temperature is generally subnormal, 95°-97° F., in a few cases it rises at the last to 102°-104° F. The breath has usually a peculiar smell, somewhat like chloroform, and acetone has been found in the expired air. This smell is often detected for some time before the onset of coma.

The acidity of the urine is increased, but the quantity of urine and the amount of sugar excreted usually diminish with the onset of comatose symptoms. The urine, like the breath, has a peculiar "chloroform" smell (acetone). A trace or small quantity of albumen is usually present (according to some writers invariably present) in the urine in the common variety of diabetic coma. Sometimes the urine is free from albumen up to the onset of comatose symptoms, but in other cases a trace of albumen is present for some time before coma develops. In 20 consecutive cases of diabetic coma the writer found a trace or small quantity of albumen in the urine in every case. According to Kulz, casts are always present in the urine in the common form of diabetic coma, and he believes the appearance of casts to be a valuable promontory sign of the onset of coma. In 17 consecutive cases of diabetic coma the writer found them always present. When a small amount of a dense yellowish-white deposit appears exactly at the bottom of the urine glass, and when this deposit consists of casts,

he believes that coma nearly always follows. The casts in diabetic coma are finely granular or hyaline, and are present in enormous numbers. The urine in diabetic coma usually gives a dark brownish-red coloration with perchloride of iron (Genhaidt's reaction). Acetone can also be detected. The amount of ammonia is much increased, and according to Stadelmann  $\beta$ -oxybutyric acid is always present. The alkalinity of the blood is said to be much decreased. The blood always decolorises an alkaline methylene blue solution as already described. The knee-jerks are usually absent (absent in 20 out of 23 cases).

The coma often becomes complete, in other cases the patient, though semi-comatose, can be roused almost up to the last. When once well-marked symptoms of coma have developed, death occurs within forty-eight hours.

The description just given is that of the common form of diabetic coma (Kussmaul's variety), but there are two rare sub-varieties. (1) *The alcoholic form*. At the onset the patient is very excited, and behaves like a drunken man. The pulse is quick, coma develops, and soon terminates fatally. Such cases have been mistaken for alcoholic intoxication. The urine gives the reaction with perchloride of iron, the breath has the "acetone" smell, but dyspnoea is absent or slight. (2) *Diabetic collapse* (described by Dreschfeld and Fierichs). The patient suddenly becomes drowsy, the extremities and face cold and livid, the pulse quick and small, and coma develops. The temperature sinks subnormal, the breath has not the "acetone" smell, and the urine does not contain acetone or diacetic acid. Dreschfeld points out that this form of coma occurs chiefly in patients over 40, and often they are well nourished or stout. Dreschfeld and Fierichs believe that the symptoms are due to cardiac failure owing to degeneration of the cardiac muscle.

*The diagnosis of diabetic coma* (in its common form) is easy, especially if the patient is known to have been suffering from diabetes for some time. If the patient be seen for the first time in the comatose state, then the diagnosis between diabetic coma and other forms of coma has to be considered. If the urine can be obtained, the presence of a large quantity of sugar, along with a high specific gravity, is sufficient to exclude most of the other causes of coma. The "acetone" smell of the breath, the perchloride of iron reaction in the urine, and the dyspnoea are also important indications in favour of diabetic coma. If the urine cannot be obtained, or if there should be any doubt as to the nature of the case, the methylene blue reaction obtained by examination of a drop of the patient's blood will definitely settle the diagnosis.

In coma from fractured skull and other

cerebral conditions occasionally the urine contains sugar, but the quantity is only small, the specific gravity is not high, there is no perchloride of iron reaction, there is no "acetone" smell of the breath and the urine, the peculiar dyspnoea is absent, and the pulse is often slow or normal, whilst in diabetic coma of the usual form the pulse is rapid, and dyspnoea is a prominent symptom. Symptoms resembling those of Kussmaul's diabetic coma are said to occur occasionally in other affections (cancer of the stomach, anaemia, kidney disease), but in such cases (as well as in the alcoholic and cardiac forms of diabetic coma) the diagnosis can be made by the urine examination or by the methylene blue reaction of a drop of the blood.

The pathological changes met with in patients dying of diabetic coma are not characteristic. The most constant are those in the renal epithelium already mentioned. In the variety of coma described as diabetic collapse, it is very probable that the cause is cardiac failure owing to degeneration of the heart muscle. In the common variety of diabetic coma, fat embolism has been suggested as a cause in certain cases. According to Schmitz, the symptoms are due to a ptomaine formed in the intestine owing to obstinate constipation. The symptoms certainly point to the action of some toxic substance. Acetone and diacetic acid have been suggested as the poison, but both substances can be given in large doses without producing coma (Drieschfeld and others). There is considerable evidence in favour of intoxication by some organic acid (Stadelmann), and  $\beta$ -oxybutyric acid or crotonic acid are thought by some to be the toxic substances. As already pointed out, the urine nearly always contains casts and a small quantity of albumen in diabetic coma, and the renal epithelium frequently presents degenerative changes. Hence it appears probable that there is impairment of the renal functions. V. Harley has shown that symptoms resembling diabetic coma can be produced in animals by injecting grape-sugar into the jugular vein and preventing its elimination by ligaturing the ureters. Now in diabetic coma the sugar elimination and urine secretion usually diminish, and from a consideration of facts mentioned it appears not improbable that coma is due to poisoning owing to the kidneys failing to eliminate certain substances produced in the diabetic organism.

**FORMS OF DIABETES AND GLYCOSURIA.** There are two chief forms besides several sub-varieties. In the *severe* form the sugar excretion is great and does not cease when carbohydrates are excluded from the diet. There is often much wasting. This form is most common in patients under middle age. In children and young persons it sometimes runs a very rapid course (a few months or even less). In the *mild* forms of diabetes the symptoms and sugar excretion

are not so marked. The sugar disappears from the urine on withdrawing the carbohydrates from the food. The patients are often above middle age, and they are not inheerently stout or gouty. The night urine contains less sugar than the day urine, or it may be free from sugar. The course is often chronic (This form is sometimes spoken of as chronic glycosuria). There are also *transitional* and *intermediate* varieties, and the mild form may pass into the severe. In some of the mildest forms thirst, dimness, and other symptoms are absent, and the only signs of disease are made out by examination of the urine, which contains sugar and has a high specific gravity (*diabetes desipens*). In another mild form the glycosuria and other symptoms disappear from time to time (*intermittent diabetes*). It has already been pointed out that glycosuria may be secondary to a number of affections (*symptomatic glycosuria*—see article "Glycosuria"). Hanot, Chauffard, and others have drawn attention to a rare disease characterised by the association of symptoms of diabetes mellitus with bronzed pigmentation of the skin, and frequently with hypertrophic cirrhosis of the liver (*diabète broncé*).

**TERMINATION.**—The severe forms always terminate fatally. The duration may be a few years (3-5), often it is shorter, occasionally it is a few months, or even less. In the mild cases life may be prolonged for many years (10 or 20). In the mild cases by restricted or rigid diet the symptoms may disappear entirely, but they usually return when an ordinary diet is taken.

In rare cases diabetes is associated with symptoms of nephritis (parenchymatous or interstitial), and the diabetic symptoms may gradually subside whilst those of nephritis remain. Also occasionally (though very rarely) the symptoms of diabetes mellitus give place to those of diabetes insipidus.

The fatal termination in diabetes mellitus is most frequently by diabetic coma. The next most frequent termination is by pulmonary phthisis. Other causes of death are carbuncle, gangrene, etc.

The **PROGNOSIS** depends chiefly on the form of the disease and the age of the patient. In the mild forms, especially if the sugar excretion can be checked by a rigid diet, and if the patient be over middle age, the prognosis is fairly favourable. But it is very grave in young persons and in the severe forms of the disease. Other unfavourable indications are marked wasting, the occurrence of pulmonary tuberculosis, a family history of the disease, unfavourable conditions of life, Gerhardt's perichloride of iron reaction in the urine, signs of commencing coma, onset of gangrene.

**Favourable indications** in addition to those already mentioned are the association of obesity

or gout, long duration of the disease without much wasting, favourable conditions of life, in the female onset about the climacteric period.

THE DIAGNOSIS of diabetes mellitus is usually easy. The disease may be overlooked, however, and the patient treated simply for one of the complications. It is important to remember that occasionally the patient complains chiefly of weakness and wasting. It is important not to mistake a temporary and mild glycosuria for true diabetes.

**PATHOLOGICAL ANATOMY**—Numerous pathological changes have been described in diabetes, yet, strictly speaking, the disease has no definite pathological anatomy. The changes met with are usually due to complications or are secondary in nature, and it would scarcely be possible for a pathologist to diagnose diabetes without a clinical history or the chemical examination of the urine or blood. For this reason the changes found post-mortem in the various organs have been already described in considering the relation of diabetes to disease of the nervous system, liver, and pancreas, and in the description of the complications. The condition of the blood has been described under the symptomatology.

**PATHOGENESIS**—The true nature of the disease remains obscure. The theories as to its exact origin are numerous, but none is altogether satisfactory. Sugar appears in the urine because there is an excess of sugar in the blood, and the symptoms of the disease are due to the same cause.

The cause of the excess of sugar in the blood has been attributed to (1) an excessive formation of sugar in the system, (2) a diminished sugar destruction, (3) by some writers it is believed that in certain cases there is an excessive sugar formation, in others a diminished sugar destruction. Bunge has given good reasons for rejecting the view of excessive formation as regards the origin of the sugar from articles of food. Kaufmann, on the other hand, has brought forward experimental evidence in favour of increased sugar formation. He isolated the liver by tying all its vessels in a healthy dog, and also in another dog which had been rendered diabetic by extirpation of the pancreas. The sugar in the blood gradually became diminished during its circulation in various parts of the body, but the blood lost the same proportion of sugar both in the healthy and in the diabetic animal. Hence, he concludes that sugar destruction was the same in each.

Many authors believe that diabetes in man is usually due to diminished sugar destruction.

In the mild forms of diabetes removal of carbohydrates from the diet causes the glycosuria to cease. In such cases the sugar appears to be derived in some way, directly or indirectly, from the carbohydrates of the food. Seegen believes that in the mild form (which he terms the

"hepatogenic") the liver cells are unable to assimilate the carbohydrates in a normal manner. But pathological anatomy has failed to reveal any definite changes in the liver associated with diabetes.

According to Pavy, the two lines of defence—intestinal villi and the liver—are inadequate to accomplish the function of synthesising the carbohydrates. Hence the latter reach the general circulation in excessive quantity and appear as sugar in the urine.

In the severe forms of diabetes the sugar in the urine is evidently not dependent simply on the carbohydrates of the food. Since the glycosuria persists when the diet consists only of fat and nitrogenous substances, and persists even when no food is taken, Pavy believes that in these severe forms the sugar is derived not only from the food but also from the tissues. He believes that the proteins of the body have a glucoside constitution, and that in diabetes of the severe form a carbohydrate is cleaved off from these proteins by the action of some ferment which he supposes to be present in the system.

According to Seegen, in the severe forms the cells and tissues of the organism have lost then function of destroying the sugar in the blood.

Limited space forbids a discussion of the subject, but whatever view we take of the sugar formation or destruction the question finally arises, *What is the cause of the abnormality in diabetes?*

Pavy believes that as regards the liver there is a vasomotor paralysis and dilatation of the small arterioles owing to some change in the nervous system. As a result the blood in the liver is in a hyper-oxygenated state, and this favours the passage of carbohydrates into glucose. But Seegen points out that Pavy was able to produce diabetes experimentally by injury to the nervous system after ligation of the hepatic artery, and he asks, therefore, how vasomotor paralysis can be regarded as the explanation.

It has been already shown that there is strong evidence that some change in the nervous system is the starting-point of the disease in many cases.

Also experiments on animals and pathological observations render it very probable that, in some cases of diabetes, pancreatic changes are the cause of the disease.

It has also been pointed out that atheroma might act as the starting-point of diabetes by producing changes in the nervous system or in the pancreas, and there are a few cases on record which afford strong evidence of diabetes being the result of pancreatic changes which were secondary to atheroma.

Diabetes has been attributed by Bunge and others to pathological chemical changes commencing in the muscles.

The opinion appears to be gradually gaining ground that diabetes is not a pathological entity, but rather a group of symptoms which may be produced by the various morbid changes already suggested. Possibly it is sometimes due to an endogenous or inherited morbid condition, and possibly to other causes.

#### TREATMENT

The results of treatment in the mild forms are good, in the severe forms very unsatisfactory. After a diagnosis has been made, before commencing treatment, the weight should be taken, complications noted, the amount of sugar estimated, and the urine tested with perchloride of iron. Then a rigid diet (practically free from carbohydrates) should be prescribed in order to ascertain whether the sugar excretion can be checked thereby. If this should occur the case belongs to the mild form of the disease, and a little carbohydrate food in the form of bread is then allowed and gradually increased (if necessary) until the glycosuria returns. The amount of bread which can be taken before the return of the glycosuria is thus an indication of the quantity of carbohydrate food the patient can tolerate. If a diet free from carbohydrates does not cause the sugar to disappear from the urine, then the patient is suffering from a severe form of the disease. If there should be great wasting, and especially if the urine should give a marked reaction with perchloride of iron, it is not advisable to keep the patient long on this very rigid diet.

Many authors think it is important in the very severe forms of the disease that the rigid test diet should not be commenced suddenly, as by such a procedure there is a possibility of producing diabetic coma (Elbstein, Naunyn, and others). A few days may be allowed to elapse in changing from a mixed to a rigid diet. Potatoes may be excluded first, then bread, and afterwards all other carbohydrates. This plan appears to the writer to be important.

Having determined the effect of diet and the form of the disease in any case, directions must be given for the future DIETETIC TREATMENT—(1) as to the *nature* of the food, (2) as to its *quantity*. The treatment must be modified in each case according to the form of the disease. A record of the weight and sugar excretion should be kept, and probably the former is the more important.

IN THE MILD FORMS OF THE DISEASE there are two classes of cases. (1) those in which the sugar excretion ceases only when carbohydrates are excluded from the diet, (2) those in which it ceases when the carbohydrate food is simply diminished in quantity. In either case the diet which is sufficient to arrest the glycosuria should be continued for several weeks. It is often found that the condition of the patient is then much improved. In the first class of cases he is

able to take a little carbohydrate, in the second class he is able to take an increased amount of carbohydrate without the glycosuria recurring. But in many of the first class of cases any addition of carbohydrates to the diet is followed by a return of the glycosuria. After a period of restriction, however, it is usually necessary to relax the diet a little, especially as regards bread, and to remain content if by a moderately restricted diet we can limit the sugar excretion to 500 or 600 grains daily. If the patient should be very stout a reduction of the total quantity of food is often of great service, but if wasted this should not be attempted.

IN THE SEVERE FORMS OF THE DISEASE, when the most rigid diet fails to remove sugar from the urine, and when there is much wasting, and especially if the urine give a marked reaction with perchloride of iron, the opinion has been gradually gaining ground for some years that a very rigid diet is injurious. In such cases, after a short period of rigid dietary (in order to determine the form of the disease), a small amount of carbohydrate food must be allowed. The diet in these severe forms should consist of nitrogenous food, fatty food, and a small amount of carbohydrate food, chiefly in the form of bread, but saccharine food should be avoided, and the carbohydrate food should only be allowed in limited quantity. Fatty food is especially important, and should be given in large quantities, a little alcohol being also allowed to aid its digestion. Cream is of great service, and should be given freely, also milk may be allowed in moderate quantities. If coma appears to be threatening the diet should be less rigid, i.e. the amount of carbohydrate should be increased. According to Elbstein, the appearance of acetone and diacetic acid in the urine is an indication for diminishing the albumen and for increasing the carbohydrates.

ARTICLES OF DIET. When a rigid diet is indicated (either for diagnosis or for treatment), the following articles should be sanctioned or forbidden—

<i>Sanctioned</i>	<i>Forbidden</i>
Butchers' meat of all kinds (except liver), potted and preserved meats	Sugar, saccharine and farmaceutic articles of food
Ham, tongue, bacon	Pasty and farmaceutic puddings
Poultry, game	
Fish (fresh, dried, and preserved), sardines, shrimps	
Broths, animal soups, and jellies (prepared without the addition of saccharine or starch materials)	Rice, sago, arrowroot, tapioca, macaroni, vermicelli, semolina
Eggs, cheese, cream	Potatoes.

Sanctioned	Forbidden
Butter, suet, oils, and fats	
Mustard (without sugar).	
Reliable bread substitutes (gluten bread, almond and aleuronat cakes)	Wheaten bread and biscuits
Green vegetables — mustard and cress, watercress, endive, lettuce, spinach, turnip-tops, cabbage, broccoli, Brussels sprouts, spring onions	Carrots, turnips, pumpkins, beetroot, beans, peas, large onions
Cucumber	Liver
Mushrooms	Oysters, cockles, mussels, the "puddings" of crabs and lobsters
Pickles (cucumber, walnuts, and onions)	Honey
Nuts (walnuts, almonds, filberts, hazel nuts, Brazil nuts), but not chestnuts.	All sweet fruit and dried fruits

## BEVERAGES

Water, soda-water, and mineral waters	Port, Tokay, champagne, and sweet wines
Tea, coffee	
Dry sherry, claret, Burgundy, hock, Moselle, Rhine wines, most Italian wines, Austrian and Hungarian table wines (all in moderate quantities, however)	Must, fruit juices and syrups
Bandy in small quantities	Sweet lemonade
	Liqueurs
	Beer, ale, porter, and stout
	Rum and sweetened gin
	Cocoa and chocolate
	Milk in large quantities

Almost all kinds of animal food may be sanctioned, but liver, oysters, cockles, mussels, crabs, and lobsters should be avoided, because they contain a large amount of carbohydrate material.

With regard to milk caution is necessary, since it contains 4 per cent of milk-sugar, but it also contains albuminous bodies and fat, which are of great service to the diabetic. In some cases the addition of milk in considerable quantity to the diet of a diabetic patient does not increase the sugar excretion, i.e. the milk-sugar is utilised in the system. In other cases the glycosuria is distinctly increased by the milk, but nevertheless such patients may gain weight. In the very severe forms of the disease milk is of great service, especially if the digestion is feeble, but in milder forms, if a rigid diet be desirable, milk should be excluded, unless it can be shown that it does not increase the glycosuria.

Cream contains less lactose than milk, but seven times the amount of fat. It may be taken

freely by diabetic patients, and is most useful in the severe forms of the disease. By the following simple method an *artificial milk* can be prepared from cream. — To about a pint of water placed in a pot or glass measure four tablespoonfuls of fresh cream are added and well mixed. The mixture is allowed to stand for twelve hours. Most of the fatty matter of the cream floats to the top, and can be skimmed off with a teaspoon, and on examination it will be found almost free from sugar (the sugar originally present in the cream having been dissolved and distributed in the water). This fatty matter is then separated and placed in a glass and mixed with water. The white of an egg is added, and the mixture well stirred. A little salt and a trace of saccharine may be added. The mixture with a little practice may be made to taste almost like milk, and it may be taken freely by all diabetic patients.

Fats are the most valuable articles of diet for diabetic patients, especially in the severe forms of the disease, and may be allowed in large quantities. The most useful are butter, cream, bacon, cheese, eggs, suet. Cod-liver oil is also of service. If fatty food should give rise to dyspepsia, a small amount of brandy and water, or other form of alcohol, taken after the meal, often aids the digestion.

Of the *carbohydrates*, starch is less injurious than sugar. Of the various kinds of sugar, glucose is most injurious. Milk-sugar and cane-sugar rank next. Lævulose is least injurious. Many observations have shown that in moderate quantities lævulose is utilised in the system, and does not increase the sugar excretion in mild forms of diabetes, but in large quantities, and in the severe forms of the disease, it is only partially utilised, and therefore the sugar excretion is increased. Saccharine and xylane may be used to sweeten articles of food in place of sugar.

When a very rigid diet is indicated fruit should be excluded, on account of the sugar which it contains. But in many kinds of fruit a large portion of the sugar is lævulose, which, as just mentioned, can be utilised in small quantities in certain cases. Hence when a very rigid diet is not necessary, a very small quantity of those fruits which contain least sugar may be allowed.

Grapes, cherries, and other fruits which contain much sugar, as well as dates, figs, currants, raisins, and other dried fruit, should be forbidden. Nuts may be allowed freely, with the exception of chestnuts. As a rule green vegetables may be allowed, whilst white vegetables and root vegetables, which contain more carbohydrate, should be avoided. These have been already indicated.

Bread is the article of diet with respect to which there is the greatest difficulty. It contains 49 per cent of carbohydrates and 2 per cent of sugar (Konig), and is thus unsuitable

when a very rigid diet is desirable for diagnosis or treatment. In such cases it must be replaced by various bread substitutes. Unfortunately many of these are very unreliable, and contain either starch or sugar in considerable quantity. Before recommending any specimen it is important to try the effect of a drop of iodine and potassium iodide solution. If the specimen becomes deep blue black it contains a large quantity of starch and is unreliable. Some bread substitutes contain sugar, and hence it is advisable to test for sugar by the fermentation test in an inverted test-tube.

In the most severe forms of the disease, when a very rigid diet is not desirable, it is best to allow a small amount of ordinary bread daily, in other cases when for diagnosis or treatment a very rigid diet is necessary, bread should be excluded from the diet for a few weeks, and some reliable substitute employed. But after a month or two most patients find bread substitutes objectionable, and it is necessary and often desirable to allow a small quantity of ordinary bread. The following bread substitutes have been employed—

(i) *Gluten Bread*—Good preparations of gluten flour are almost unaffected by iodine, and contain only 2 to 3 per cent of carbohydrate. The flour can be purchased and the bread prepared at the patient's house according to the directions supplied by various firms. With care a fairly palatable and reliable preparation may be obtained which will be of service as a bread substitute, and will cause a reduction of the sugar excretion. But many preparations are very unpalatable and unreliable. (ii) *Bran cake* was recommended by Campbell many years ago, but is now seldom used. (iii) *Soya biscuit* and bread have been largely used recently. They are prepared from soya beans, and some specimens contain only a small percentage of starch (3 to 6), but others contain as much as 30 or 45, and are therefore useless. (iv) *Almond cakes* were recommended by Pavy long ago. They can be prepared at the patient's home from almond flour. About 4 ounces of almond flour are mixed into a paste with a little water and German yeast. The mixture is allowed to stand in a warm place for about twenty minutes. Then one egg, beaten up, and a little cream and water are added, and the whole is mixed into a paste, divided into cakes, and baked for fifteen to thirty minutes. (v) *Cocoa-nut cakes* may be prepared in the same way by using desiccated cocoa-nut powder in place of almond flour. Both almond flour and cocoa-nut powder contain a small amount of sugar, which is destroyed by the action of the yeast used in making the cakes. Both of these cakes contain a large amount of fat, and hence a little alcohol is often necessary to aid their digestion. Almond flour and cocoa-nut powder may be used for the preparation of pudding.

(vi) *Aleuron* is a vegetable albuminous substance which contains only a very small percentage of carbohydrate, and which has been strongly recommended by Elstein. The writer has found that palatable and reliable biscuits can be prepared by mixing aleuronat and cocoa-nut powder.

Two ounces of desiccated cocoa-nut powder are mixed with a little water and German yeast, so as to form a paste. This is kept in a warm place for half an hour, then 2 ounces of aleuronat, one egg (beaten up), and a small quantity of water, with a little saccharine, are added. The whole is mixed into a paste, which is spread out on a tin, divided into cakes, and baked for twenty or thirty minutes.

THE BEVERAGES which are free, or almost free, from carbohydrates may be allowed, and they have been already indicated. Alcohol does not increase the sugar excretion, and therefore alcoholic drinks which contain only a very small quantity of carbohydrate may be allowed in moderation. In the severe forms of the disease, alcohol is of service in enabling the patient to take large quantities of fatty food without dyspeptic troubles.

The following is a useful lemonade for the relief of thirst—citric acid 10 grains, glycerine 1 drachm, water 1 pint, this may be taken in small quantities during the twenty-four hours, or a lemonade may be made from fresh lemons and sweetened with saccharine. Other acid drinks may be used. The largest quantity of fluid should be taken before, not after, a meal.

THE MODE OF LIFE AND GENERAL HYGIENIC CONDITIONS.—It is important to relieve the patient from mental worry and anxiety as much as possible. Considerable muscular exercise in the open air is of great service in some of the milder cases, and sometimes causes a diminution of the glycosuria, but in the severe forms it is injurious. Massage is of service in some cases (according to Grube in those associated with arterio-sclerosis).

TREATMENT BY ALKALINE MINERAL WATERS.—The spas of Carlsbad, Marienbad, Neuenahr, and Vichy are much frequented by diabetic patients on account of the supposed virtues of their mineral waters. The salts in the waters of Carlsbad and Marienbad consist chiefly of sodium sulphate and sodium bicarbonate, those in the waters of Neuenahr and Vichy chiefly of sodium bicarbonate. It is useless and often dangerous to send English patients suffering from the severe forms of diabetes to these spas. The long journey has not infrequently acted as an exciting cause of diabetic coma. Arterio-sclerosis is stated by Schmitz to be a contraindication of the Neuenahr waters. It is certain, however, that patients suffering from the mild forms of the disease (chiefly those which are associated with obesity or gout) do

often derive much benefit from a visit to these spas. But whether the results are due to the action of the waters or to the improved general conditions of life, more careful diet, and life in the open air, is a disputed point. (But from limited observations at Carlsbad and Neuenahr, the writer is inclined to attribute part of the good results obtained in the mild cases to the action of the waters.)

**MEDICAL TREATMENT**—It is certain that no drug hitherto tried has a definite curative action, but several have a beneficial effect.

*Opium and its alkaloids* have been long employed, and are probably more useful than other drugs. At first half a grain of opium may be given three times a day. The dose may be gradually increased to 2 or 3 or more grains of opium three times a day, as diabetic patients are very tolerant of the drug. Ralfe advised it to be given an hour after a meal, since at this time he believed it had a greater effect in restraining diuresis, and was less liable to cause dyspeptic symptoms. Some authors prefer the crude opium, others morphia, and others codeia. The last drug has been specially recommended by Pavy. It may be given in small doses at first, half a grain three times a day, and gradually increased to 2 or 3 grains. All the opium preparations are liable to cause troublesome constipation and dyspeptic symptoms, and therefore require watching, but probably codeia is less injurious in this respect.

Next to opium and its alkaloid, *alkalies* have been, perhaps, most frequently employed in the treatment of diabetes. The alkalies which are most often used are the bicarbonate of soda, and the citrate, acetate, carbonate or bicarbonate of potash.

Naunyn strongly advocates the use of sodium bicarbonate in large doses in severe cases of diabetes before the onset of symptoms of coma, and when there are indications of acid intoxication, such as great excretion of ammonia or marked reaction of the urine with perchloride of iron. So long as the perchloride of iron reaction is intense, he advises that bicarbonate of soda (150 to 450 grains daily) should be given, and that it should be increased in amount if the perchloride reaction increases. Since adopting this treatment (twelve years ago) the number of cases of coma in his practice has diminished greatly.

*Lithium* salts (carbonate and citrate) have been much employed (often combined with arsenic). In cases associated with gout they may be of service.

*Arsenic* has been much prescribed in the treatment of diabetes, and in some of the milder cases it probably is of some service, when the dose is gradually increased (up to 10 m of the liquor arsenicalis three times a day).

*Jambul* has been often prescribed in diabetes during recent years. Many observers have

found it useless, others have obtained favourable results when the drug has been given in sufficiently large doses.

*Sodium salicylate* has been recommended by Ebstein, who prescribes it freely. Brunton and Ralfe think it is of most service in the glycosuria of gouty persons. Schmitz of Neuenahr obtained better results with *salicylate of bismuth* than with any other drug in the mild forms of the disease ( $7\frac{1}{2}$  grains twice a day).

Recently *uranium nitrate* has been recommended by West, and there is certainly some evidence in its favour.

Numerous other drugs have been recommended from time to time, but usually extended experience has shown them to be useless or of very slight value.

*Cod-liver oil* or *lipanin* are to be recommended when the patient is much wasted.

**TREATMENT OF COMPLICATIONS**—When irritation and eczema of the genital organs (vulva or prepuce) is a troublesome symptom, it is important that the external orifice of the urethra and surrounding parts should be dried with lint or absorbent cotton-wool directly after each act of micturition. Boracic acid ointment, boracic acid lotion, or a solution of sodium hyposulphite (one in forty) may be applied. The treatment of other complications—boils, carbuncles, gangrene, phthisis, nephritis, etc.—is described elsewhere in the articles devoted to these subjects.

**PREVENTION AND TREATMENT OF DIABETIC COMA**—In the severe forms of diabetes, when there is much wasting or when the urine gives a marked reaction with perchloride of iron, there is great danger of coma developing. In such cases it is important to remember that a long railway journey, over-exertion, or sudden change of diet is liable to be followed by coma. Prolonged constipation is also dangerous. As already mentioned, the diet ought not to be too rigid. Some carbohydrate food (especially bread) ought to be allowed. Cream and fatty food should be given freely. Whenever there are signs of commencing coma, the carbohydrates in the diet should be increased a little and the nitrogenous food diminished. Constipation should be relieved by the use of purgatives, and Schmitz believes that he has checked the onset of coma by producing free action of the bowels with castor oil.

In severe cases, especially when the perchloride of iron reaction is intense, alkalies should be given in large quantities—400 to 500 grains of sodium bicarbonate in the twenty-four hours. Naunyn believes that this treatment is of great importance, and that the onset of coma may sometimes be prevented thereby for a long period. When early symptoms of coma are observed alkaline treatment ought to be commenced at once, if it has not been prescribed previously. A number of cases are now on



record in which the early symptoms of coma have subsided under vigorous alkaline treatment

Sodium bicarbonate may be given in a little milk or in an effervescent draught with a little citric acid and saccharine (Yeo), or citrate of sodium may be given (900 grains in the twenty-four hours, Lépine)

During the last ten years the intravenous or subcutaneous injections of warm alkaline or salt solutions, as recommended by Stadelmann, have often been tried when coma has become well marked. There can be no doubt (1) that this method of treatment has often a decidedly beneficial effect, but (2) that the results are usually only temporary, and a fatal termination is very rarely prevented.

Solutions which have been chiefly used are A 3 to 5 per cent solution of sodium bicarbonate in 0.6 to 0.7 per cent sodium chloride solution, a 3 per cent watery solution of sodium bicarbonate, a solution of 10 grammes of sodium bicarbonate and 7 grammes of sodium chloride in a litre of sterilised water (Lépine), a 0.6 per cent solution of sodium chloride (v Noorden). Intravenous injection (into the median basilic vein) is to be preferred to subcutaneous injection. It is important that the fluid should be warm (Lépine recommends a temperature of  $38^{\circ}\text{C} = 100.4^{\circ}\text{F}$ ). A large quantity of fluid should be used (2½ pints Oliver, 2 litres Lépine).

Usually the only result is an improvement in the pulse and a diminution of the coldness of the skin and a little diminution of the coma, so that the patient can be more easily roused. But occasionally the coma disappears partially or completely, so that the patient can converse with his friends, usually a relapse soon occurs.

**Diacetic Acid.**—Diacetic acid ( $\text{CH}_3\text{COCH}_2\text{COOH}$ ) or aceto-acetic acid is sometimes found in the urine in diabetes (it is probably never present in health), with a solution of chloride of iron it gives a Bordeaux or Burgundy red colour, but not if the urine has been previously boiled and allowed to cool (p. 326), it breaks up quickly into acetone and carbonic acid. See DIABETES MELLITUS (*Diabetic Coma*), URINE, PATHOLOGICAL CHANGES IN (*Acetone, Acetoacetic Acid*, etc.)

**Diaceturia.**—The presence of diacetic acid in the urine, Gerhardt's reaction (see p. 326). See UNCONSCIOUSNESS (*Auto-intoxications*)

**Diacetylmorphine.** See HEROIN

**Diachalasma.**—A fissure or cleft (from Gr διαχάλαω, I loosen, and διαχάλασμα, a hiatus)

**Diachoresis.**—The excretion of faeces (from Gr διαχρώω, I pass through)

**Diachrosis.**—An eruption (from Gr διαχρίω, I anoint).

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**Diachylon.**—Emplastrum plumbi or lead oleate, diachylon ointment is formed by melting together equal parts of lead plaster and soft paraffin, and mixing with an equal quantity of zinc oleate ointment and mercuric oleate ointment, diachylon pills have been used to provoke abortion.

**Diaderm.**—A blastoderm, in which there are two layers or plates of cells, ectoderm and entoderm, joined at their edge (ectental line), and surrounding a central segmentation cavity (*Microt*), the earliest form of the diaderm is known as the *blastula*.

**Diaeresis.**—Separation or solution of continuity. In the classification of teratology, Taruff grouped the monochorionic twins, the placental parasites, and the united twins under the heading of the disomita, to the monochorionic twins and placental parasites he gave the name *dioretic disomata*, for the bodies of the two fetuses are separate, although there may be communication by means of the vessels of the umbilical cords, the united twins he called *synergetic disomata* (Ballantyne's *Antenatal Pathology*, vol II p. 623).

**Diagnosis.**—The process of distinguishing between different states, usually different diseases, it implies a certain amount of difficulty, and when difficulty is absent the process is one rather of recognition than of diagnosis. *Symptomatic* diagnosis depends upon the consideration of symptoms alone, while *physical* diagnosis is based on the physical signs which may be elicited by the medical man. It is almost tautological to speak of *differential* diagnosis. *Diagnosis by exclusion* is the process by which the presence of every other possible state has been shown by the conditions present to be insufficiently established, the only other possibility that is left is then regarded as the state which is present. See ABDOMEN, CLINICAL INVESTIGATION OF, GYNECOLOGY, DIAGNOSIS IN, POST-MORTEM METHODS, etc, etc.

**Diagonal Conjugate.** See LABOUR, PHYSIOLOGY OF (*Hard Passages, Diameters*)

**Dialuric Acid.**—A monobasic acid ( $\text{C}_4\text{H}_4\text{N}_2\text{O}_4$ ) obtained from alloxan ( $\text{C}_4\text{H}_2\text{N}_2\text{O}_4 + 4\text{H}_2\text{O}$ ) by the action of hydrogen sulphide.

**Dialysed Iron.**—Liquor Ferri Dialysatus (*Wyeth*) is a mild preparation of iron, recommended in the case of delicate children, and given in doses of 5 to 30 m. in water.

**Dialysis.**—The interdiffusion of two liquids (one of which is generally pure water) separated from each other by an animal membrane or parchment paper, the portion of a liquid which passes through into the water is called the *diffusate*, and the portion left behind is the *dialysate*, crystalloids diffuse much more quickly than colloids.

**Diameter.**—The distance from a point in the periphery of a circle passing through the centre to the corresponding point on the opposite side; the same measurement made in the case of a spherical, circular, or cylindrical body. There are various pelvic diameters (such as the conjugate, true, diagonal, and external), the cranial diameters (such as the occipito-frontal, occipito-mental, biparietal, etc.), and the corporeal diameters (such as the bisacromial, the sterno-vertebral, etc.).

**Diamido Acids.**—Acids with two amidogens ( $\text{NH}_2$ ) replacing two hydrogen atoms in their molecule, such as *lysine* (diamido-caproic acid) and diamido-propionic acid.

**Diamines.**—Bases in which two atoms of hydrogen in ammonia have been replaced by radicals, e.g. *ethylene-diamine* ( $\text{C}_2\text{H}_4(\text{NH}_2)_2$ ), and *diethylene-diamine* or *piperazine* ( $\text{NH}(\text{C}_2\text{H}_4)_2\text{NH}$ ). See URINE, PATHOLOGICAL CHANGES IN (*Abnormal Nitrogenous Constituents, Diamines*).

**Diaminuria.**—The presence of diamines (cadaverine, putrescine, etc.) in the urine. See URINE, PATHOLOGICAL CHANGES IN (*Abnormal Nitrogenous Constituents, Diamines*).

**Diapedesis.**—The passage (during inflammation) of the red corpuscles of the blood out of blood-vessels into the surrounding tissues; they are supposed to pass through the unruptured vessel walls either between the endothelial cells or through openings made by the leucocytes, but it is more likely that they pass out through the bursting of capillaries; the process is to be distinguished from the emigration of leucocytes, occurring as a rule before the latter has become active. See PHYSIOLOGY, NUTRITION OF THE TISSUES, THE BLOOD (*Cells of the Blood*); SUPPURATION (*Acute Circumscribed Abscess*).

**Diaper.**—A napkin, worn to absorb discharges from bladder, bowels, or vagina; sanitary towel.

**Diaphoretics.** See also PHARMACOLOGY; PRESCRIBING; AMMONIUM; etc.—The term diaphoretics is applied to all measures which increase the secretion of sweat.

*The Physiology of Sweating.*—The activity of the sweat glands is closely related to the condition of the cutaneous circulation, and therefore to the heat-regulating mechanism. When the surrounding atmosphere is warm the cutaneous vessels dilate, the amount of sweat is increased, and heat is lost by the resulting evaporation. When the atmosphere is cold the vessels of the skin contract and the amount of sweat is decreased. The sweat glands are not, however, actually dependent on the cutaneous circulation, but their activity is under the influence of a nervous mechanism analogous to that presiding

over other secreting organs. This fact is supported not only by experimental evidence, but by such evidence as the occurrence of sweating from mental emotion, the sweating brought about by a venous condition of the blood, or the reflex sweating which results from the introduction of pungent substances into the mouth.

*Enumeration and Mode of Action.*—The principal diaphoretics are warmth; hot drinks; *jaborandi* (*pilocarpin*); liquor ammoniæ acetatis or citratis; potassii citras; potassii nitras; *ipeacacuanha* (*Dover's powder*); opium; antimonium (*pulvis antimonialis* or *vinum antimoniale*); alcohol; salicin and the salicylates; various pungent and aromatic substances.

We find the simplest means of producing diaphoresis in the direct application of heat to the skin, whether by means of warm baths, of hot air or vapour baths, or the various kinds of packs. The sweating appears to be brought about not by direct stimulation of the sudoriferous glands, but through the action of the central nervous system. It may be greatly aided by the administration of hot drinks, possibly owing to an increased flow of warm blood through the nerve centres; and these, further, may be stimulated reflexly by the addition to the drinks of pungent or spicy substances. Alcohol produces diaphoresis chiefly by dilating the cutaneous vessels and increasing the circulation through the skin, but it also stimulates the nervous mechanism. Like other narcotics, such as opium, it produces sweating in the later stages of its action by increasing the velocity of the blood. *Pilocarpin* produces copious sweating chiefly by stimulating the peripheral ends of the secretory nerves, while nicotine produces a similar result chiefly by acting on the central nervous system.

*Uses.*—Diaphoretic measures are largely used on account of their antipyretic effect in pyrexia associated with the onset of acute specific fevers, or the occurrence of acute local inflammations. For example, at the onset of an attack of acute nasal or bronchial catarrh the administration at bedtime of a small dose of opium combined with other diaphoretics will often be found to greatly relieve both the local and the general discomfort. A quarter of a grain of morphia may be ordered in a glass of hot toddy, or from 5 to 10 grains each of *Dover's powder* and phenacetin. This should be followed on the next day by the administration of such diaphoretics as liquor ammoniæ acetatis and spiritus ætheris nitrosi; antimony, *ipeacacuanha*, and occasionally aconite are useful in similar cases.

In some skin diseases associated with deficient activity of the sweat glands, diaphoretics are of assistance in the treatment.

Perhaps the most important use of diaphoretics has arisen from the recognition of the fact that the secretory action of the skin may be to

a certain extent vicarious. Hence the value of diaphoretic measures in acute or chronic nephritis with suppression of urine or indications of the supervenition of uræmic symptoms. Here the use of the hot mustard pack or the hot vapour baths, with such adjuvant measures as have been already mentioned, may be sufficient, but in many cases the powerful aid of pilocarpin is called for and proves of the greatest value. It is best administered in small doses hypodermically, the patient meanwhile being warmly covered in bed. If there is any weakness of the heart, stimulants (sal volatile) should at the same time be administered internally.

## Diaphragm.

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See APNŌYXIA (*Artificial Respiration, Depression of Diaphragm*), ANŌMA (*Symptoms, Nature and Etiology, Spasm of the Diaphragm*), CHEST, INJURIES OF (*Wounds, Perforation of the Diaphragm*), Hiccough, HYDATID DISEASE (*Echinococcus of Liver, Displacement of Diaphragm*), LUNG, TUBERCULOSIS (*Diagnosis, Röntgen-Ray Illumination, Diaphragmatic Movement*), MUSCLES, DISEASES OF (*Trichinæ, Impeded Action of Diaphragm*), POST-MORTEM METHODS (*Examination of Thorax and Abdomen*), SPASM (*Hiccough*), STOMACH AND DUODENUM, DISEASES (*Sub-phrenic Abscess*).

THE Diaphragm, or Midriff, the partition between the thorax and the abdomen, is a double muscle, the two sides of which, though capable of independent contraction, act in health habitually together. Its muscular fibres arise from the lumbar vertebrae by means of the crura, from the arched ligaments, from the cartilages of the six lower ribs, and from the posterior surface of the ensiform cartilage. Arching upwards and inwards these fibres converge on the tendon in the centre. Over the lower surface of the diaphragm is spread the peritoneum, over its upper the pleurae and pericardium. It is perforated in various places, and thus allows of the passage through it of the aorta, the œsophagus, the vena cava, the splanchnics, the sympathetic, etc. The diaphragm is supplied mainly by the phrenic nerves (each nerve controlling one lateral half), and perhaps to some small extent by the lower intercostal nerves.

During inspiration the two lateral halves of the diaphragm contract simultaneously, thus diminishing the curve of the arch and increasing the capacity of the thorax. The type of breathing in women being chiefly costal, the diaphragm is not so much used by them, and its excursions are not so well marked as is the case in men.

Apart from the results of disease of the

phrenic nerves, the height at which the diaphragm stands is dependent on the relation of the pressure in the thorax to that in the abdomen. When this relation is disturbed the diaphragm is pressed upwards or downwards according as the balance of pressure is greater on the abdominal or on the thoracic side. But as this displacement is of interest, not from the point of view of the diaphragm, but from that of the viscera which are affected, the subject will not be considered here. Nor shall we do more than mention that variety of pleurisy in which the pleural covering of the diaphragm is affected. The same remark applies to peritonitis in which the peritoneal covering of the diaphragm is implicated. In both cases the muscular fibres may show some degree of inflammation.

No tumours originate primarily in the diaphragm, but that structure is often the seat of secondary growths. Tuberculous and malignant processes are apt to pass from the peritoneum through the diaphragm to the pleura, more usually, I think, on the right side of the body than on the left. The muscular fibre of the diaphragm is liable, like other striated muscle, to suffer from fatty infiltration and from fatty and hyaline degeneration, and most probably rheumatism may also attack it. Gastric ulcers, in which the peritoneal surface of the stomach, having become inflamed, has attached itself firmly to the serous surface of the diaphragm, may make their way through that structure.

The main interest of the diaphragm from a medical point of view lies in the fact that it is an index to the state of the phrenic nerve and its centre. Under one set of conditions you may have paralysis, under another spasm.

**PARALYSIS.**—Apart from that paralysis sometimes seen in hysteria, and which is usually of little moment, the muscle may be paralysed by any lesion of the phrenic nerve or of its centre. Whether the paralysis affects one or both sides of the diaphragm depends, of course, on whether one or both phrenic nerves are implicated. Traumatic lesions of the upper cervical vertebrae, tuberculous processes there, tumours, inflammatory or other swelling of the meninges, may so interfere with the phrenic nerves as to stop the conduction of motor impulses and cause paralysis of the diaphragm. In their long and deep course through the neck these nerves, though protected from ordinary injury, may be divided in cases of wounding, or seriously compressed by tumours. Even in their course through the thorax the phrenic nerves sometimes suffer from the pressure of mediastinal tumours or are involved in inflammatory processes.

But, more commonly, paralysis of the diaphragm is due to some affection of the whole neurion. Any of the poisons, known and unknown, which produce neuritis may affect the phrenic nerve. Paralysis of the diaphragm is

believed to arise from rheumatism, though this must be a rare occurrence. It is seen more commonly in diphtheria, in alcoholic neuritis, and in beri-beri, and the toxin of influenza may produce like effects. The phrenic nerves may be involved in cases of lead-poisoning, and in progressive muscular atrophy, amyotrophic sclerosis, and bulbar paralysis, the motor cells of the phrenic nerves are sometimes affected and the diaphragm in this way paralysed. In cases in which the pleural or the peritoneal surfaces covering the diaphragm are inflamed, a certain degree of paralysis results. To some extent the defective movement of the diaphragm in these cases is no doubt voluntary, and owing to the pain which movement occasions, it is probably also in part reflex. But it may be due in some measure to the inflammatory process penetrating from the serous covering to the substance of the diaphragm, and there involving the small nerve branches in such a way as to cause paralysis.

**Symptoms**—When both sides of the diaphragm are paralysed there is no longer to be observed the normal protrusion of the epigastrium on inspiration, indeed there is sometimes a sinking in in that region. During expiration, on the other hand, the epigastrium protrudes. When only one phrenic nerve is paralysed these signs are unilateral. So long as the patient lies perfectly still there is little or no dyspnoea, though the rate of breathing is somewhat accelerated. But on the least exertion dyspnoea at once shows itself.

Litten has recently pointed out that the normal movements of the diaphragm may be readily recognised, especially in men, if the thorax be carefully watched about the sixth intercostal space. As the diaphragm separates itself from the thoracic wall a shadow moves downwards in a wave-like fashion over two or three interspaces, and again returns during expiration. To recognise this phenomenon the patient should be placed in a recumbent posture with the feet towards a good light. The observer should stand about three feet off with his back to the light. This sign might be of great importance in investigating doubtful cases.

Save in hysterical cases diaphragmatic paralysis is always of grave import, because any pulmonary complication, even of a light nature, is thereby rendered dangerous.

The *treatment* is that of the malady in the course of which the diaphragmatic paralysis has appeared. Blisters or hot applications over the course of the phrenic nerve in the neck are said to do good. Occasionally stimulation of these nerves by means of the faradic or of the galvanic current may be beneficial.

**SPASM** of the diaphragm may take either the tonic or the clonic form. Of these the latter is the more common.

*Tonic spasm* of the diaphragm occurs in

bronchial asthma. The centripetal impulse passing up the vagus to the respiratory centre is reflected down the phrenic nerve, and so causes a tonic contraction of the diaphragm, a condition clinically recognisable by lowering of the inferior borders of the lungs. Apart from that occurring in asthma, tonic spasm of the diaphragm is an exceedingly rare affection. It has been known to accompany rheumatism, and to occur in cases of tetanus and tetany. When the diaphragm passes into tonic contraction the symptoms are very striking. The lower thorax expands, the epigastrium protrudes, the liver is pressed down, and the lung border is lowered. Along with these signs there develops an extreme and dangerous dyspnoea. To counteract the descent of the diaphragm the abdominal muscles contract powerfully, and to diminish the dyspnoea the extraordinary muscles of respiration come into vigorous action. The patient also complains of severe pain round the thorax at the level of the diaphragmatic attachment.

The condition is a serious one, and may prove fatal. The treatment must, therefore, be energetic and rapidly carried out. Some relief may be obtained by means of fomentations and sinapisms, and in a hot bath the spasm may relax. Duchenne advised the use of the faradic brush over the skin in the neighbourhood of the diaphragm. The administration of the nitrates, of chloroform, or of a morphia injection may be rapidly beneficial.

*Clonic contraction* of the diaphragm, commonly known as hiccough, is much more common than tonic spasm. The contractions, which are usually fairly regular, vary much in rate, being sometimes as slow as four or five in the minute, and sometimes as frequent as one hundred. The spasm is not usually limited to the diaphragm. The glottis is also narrowed and the nares move. Such constantly recurring diaphragmatic contraction has of necessity a considerable effect on respiration. If the rate of the hiccough is rapid, there may be considerable dyspnoea, and even the act of swallowing may be interfered with. The patient is shaken by each contraction, there is considerable pain along the diaphragmatic attachment, and much exhaustion may result. If the attack be prolonged—and some last for weeks—the condition may prove very serious.

Hiccough is due to irritation of the respiratory centres, and this irritation may be peripheral or central. The *peripheral causes*, which are the commoner, include such sources of irritation in the alimentary tract as are produced by sticture of the œsophagus, by overloading of the stomach, by gastric or intestinal affections, by appendicitis, by peritonitis, and by affections of the liver. Disease of the bladder, of the uterus, or of the prostate may, in the same reflex way, induce hiccough. Irritation of the pleura or of the pericardium may act in a similar fashion. *Central causes* are seen in cases arising from cerebral

or spinal lesions, and in those which follow strong mental emotions, such as fear or anger. Poisoning of the nerve centres is probably the method of origin of that hiccough which shows itself in such diseases as typhoid fever, septicæmia, and uræmia, and of the form which so frequently precedes the lethal exit. It is to be remembered that in most cases of hiccough a neurotic predisposition is present.

As to *treatment*, it is clear that efforts should be made to remove the cause, if that can be recognised. But, apart from this, it will often be found that other forms of excitation of the nerve centres may bring the attack to an end. Sneezing may do so, for example, or coughing, or the exhibition of an emetic. Holding breath after inspiration, and making a strong expiratory effort while the glottis is kept closed, may be of benefit. The writer has seen steady pressure over the phrenics do good in an obstinate case of hiccough. Galvanisation of these nerves should also be tried, the positive pole being applied over the nape of the neck, and the negative over the phrenic. Hot fomentations applied round the lower thorax and over the epigastrium sometimes do good, or counter-irritation may be tried over these areas with benefit. In serious cases it may be needful to resort to morphia, to atropine, to chloroform, or to nitrates.

### Diaphragm, Surgical Affections of the.

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**SURGICAL ANATOMY**—The diaphragm is a large dome-shaped musculo-tendinous structure separating the thoracic and abdominal cavities. Its muscular elements take origin below from the bodies of the upper three or four lumbar vertebrae and their intervening discs, from the arched ligaments which extend thence to the transverse processes and tip of the last rib, from the cartilages of the lower six ribs, and from the back of the sternum. They converge to be inserted into the strong central tendon, which is the highest portion of the diaphragm, lying about the level of the lower edge of the sternum, or of the seventh chondro-sternal articulation. The vault of the diaphragm reaches to the level of the fifth rib on the right side, and not quite so high on the left. Its nervous supply is derived from the phrenic and sympathetic nerves.

On the thoracic side the pleura and pericardium overlie the diaphragm, while the peritoneum lines its abdominal aspect.

The continuity of structure of the diaphragm

is interrupted by three large openings for the transmission of the aorta, the inferior vena cava, and the œsophagus, and by smaller foramina for the vena azygos minor, the splanchnic, and the sympathetic nerves. Any one of these openings may be unduly large and so predispose to hernia, that for the œsophagus being the most important in this connection. As bearing upon this point also, as well as on the question of perforation of the diaphragm by inflammatory products, Tillmann has drawn attention to the fact that at certain points the muscular tissue is often deficient, so that the pleura and peritoneum alone keep up the integrity of the arch. Although these gaps vary greatly in size, shape, and situation in different individuals, their presence is fairly constant, the most important being (1) that between the spinal and costal origins of the muscle, in relation to which are the kidney, with its surrounding connective tissue, and the liver, (2) that between the costal and sternal fibres, over which the pleura lies on the right side, and the pericardium on the left, (3) sometimes the sternal fibres are entirely wanting, so that a wide space is left, over which lies the anterior mediastinum.

In some cases the lower part of the diaphragm is exceedingly thin, more rarely one half or even the whole of the muscle is wanting.

**INJURIES**—Although traumatic lesions of the diaphragm are by no means uncommon, they seldom occur apart from injuries of the viscera which lie in its immediate vicinity, and it is the signs of damage to the thoracic or abdominal organs which dominate both the pathological and the clinical picture in these cases.

*Open wounds* are usually the result of gunshot injuries, punctures, or sabre cuts. As the lung does not extend down as far as the pleura, or the pleura as far as the diaphragm, it is possible for the diaphragm to be wounded alone, or along with the pleura, while the lung escapes.

*Subcutaneous rupture* is commonest after falls or crushes, but may result from severe muscular efforts, such as those of vomiting or parturition. The jagged end of a fractured rib, or even an unskilfully manipulated œsophageal bougie, has been known to perforate the diaphragm.

As this muscle never is, and never can be, at rest, its wounds either fail to close, or heal with a thin, weak, and stretchable cicatrix, which readily favours the formation of a hernial protrusion.

**Clinical Features**—So far as the symptoms of injury to the diaphragm itself can be dissociated from those of the concomitant visceral lesions, they would appear to be *localised pain*, which is aggravated on deep inspiration or coughing, and which leads to the patient restraining the action of the muscle as far as possible. *Bleeding* may take place from open wounds either externally or into the thoracic or

abdominal cavities. Shock may be so severe as to end fatally *Rhus ardensus*, which is supposed to be peculiarly related to morbid conditions affecting the diaphragm, may be present.

As a matter of fact, however, the diagnosis of an uncomplicated injury to the diaphragm is exceedingly difficult, and in the case of simultaneous rupture all but impossible.

The treatment can only be discussed along with that of the associated lesions of the lung, pleura, pericardium, stomach, etc (*q v*). One of the great risks attending such injuries is the strangulation of any portion of the alimentary canal which may become herniated, either at the time of the accident or long afterwards. So real is this danger that Stephen Paget advocates a systematic attempt being made to close such ruptures by introducing sutures from the thoracic side of the rent. The mortality of wounds of the diaphragm is exceedingly high (29 out of 33 cases ending fatally according to Frey), peritonitis, empyema, shock, or hemorrhage being the commonest cause of death.

**DIAPHRAGMATIC HERNIA**—Any protrusion through the diaphragm is spoken of as a diaphragmatic hernia, although in the vast majority of cases—about 88 per cent—there is no hernial sac, and the condition is rather one of prolapse than of true hernia.

**Morbid Anatomy**—The protrusion may take place (1) through one or other of the natural openings in the diaphragm, particularly that for the œsophagus, (2) through one of the congenital deficiencies in the muscle, described by Tillmann, (3) through a rupture produced by indirect violence or muscular effort, or (4) through a direct wound of the muscle.

Lacher, to whose researches we owe much of our knowledge of this subject, found that in 276 cases collected by him, only 28 presented a sac, and of these 25 were congenital, and in Bohn's collection of 80 congenital cases only 14 had a sac. In round numbers, therefore, about 20 per cent of congenital cases have a sac, and are therefore true hernia, while only about 2 per cent of traumatic cases can be so described.

That the great proportion of diaphragmatic protrusions, whether with or without a sac, should be situated on the left side is natural, when we consider the substantial support given to the opposite side by the liver, the fact that all the large anatomical openings as well as the congenital deficiencies are towards the left side, and that most suicidal and homicidal wounds are aimed at the heart.

Of 150 traumatic cases, 127 were left-sided, and of 117 of congenital origin, 98 were on that side.

The greater liability of men to accidental and other forms of injury may explain the fact that diaphragmatic hernia is five times commoner in the male sex than in females.

The opening is usually far back through the tendinous part of the diaphragm, but may be met with elsewhere, particularly through the opening for the œsophagus. It varies in size and shape from a mere slit or rounded aperture to a complete absence of one half of the muscle.

As a rule, portions of more than one viscous are prolapsed. The relative order of frequency may be gathered from the following—Stomach, 151 times, colon, 145 times, small intestine, 83 times, liver, 45 times, duodenum, 35 times, pancreas, 27 times, œcenum, 20 times, kidney, 2 times.

It is interesting to observe, from the point of view of operation, that adhesions between the different prolapsed viscera or to the diaphragm seldom occur.

**Clinical Features**—(Children born with a congenital diaphragmatic hernia seldom live long enough to manifest symptoms which lead to a diagnosis. If not still-born, they usually survive but a few hours or days, and the abnormality is only discovered on post-mortem examination.)

On the other hand, it is not uncommon for a large diaphragmatic hernia to be found at the autopsy on a person who has never manifested any of the ordinary clinical features of such a condition. In all cases, moreover, the diagnosis is one of extreme difficulty, and appears to have been made with accuracy only in some seven out of nearly three hundred cases.

The prominent features in well-marked cases of some standing are (1) an unnatural depression in the upper part of the abdomen, with a corresponding fullness in the lower thoracic region. (2) Well-marked signs of air in the pleural cavity, simulating those of pneumothorax, are present, varying in extent and degree with the amount of the alimentary tract which has been displaced, and the particular viscera involved. Leichtenstein has suggested that the amount of stomach in the hernia may be estimated by filling it with water or air from the mouth, or the colon from the rectum. Distinct intestinal gurgling may sometimes be heard on auscultation of the chest. (3) The heart may be displaced and its action interfered with, causing palpitation, attacks of dyspnea, and a feeling of oppression in the chest, with inability to lie on the affected side and cough. These phenomena also vary in intensity with the size and nature of the herniated viscera. (4) Vague and atypical symptoms of dyspepsia are prominent features of all these cases. Pain in the epigastrium, heartburn, nausea, vomiting, thirst, or constipation alternating with diarrhoea are commonly complained of. Sometimes these symptoms are worst after exertion, occasionally a full meal gives temporary relief, and in some instances the patient is conscious that the food lodges in the region of the chest, where it produces a fixed pain.

When the hernia is *suddenly developed* the predominant clinical feature is intense dyspnoea with severe precordial pain, a sense of oppression, and an inability to lie on the affected side, irritative cough, and a sensation that something has given way. Shock is marked, and may prove fatal, or death may result from compression of the lung.

A diaphragmatic hernia is liable to become *strangulated* either at the time of its production or at any time thereafter. Some violent muscular effort usually determines this event, but in some cases it has been inexplicable. The patient presents all the clinical features of acute intestinal obstruction, without, as a rule, any guiding symptom to indicate the seat of strangulation. Hence the condition is seldom recognised during life. Rupture of gangrenous bowel into the pleural cavity will give rise to an empyema, and a cure may follow its evacuation by thoracotomy.

*Treatment*—An established hernia without symptoms of strangulation is so seldom diagnosed that the question of deliberate surgical intervention scarcely arises. It is only in cases of recent wounds of the diaphragm, with protrusion of viscera, and in cases of strangulation, that treatment is possible, and then it is only by operation that any good can be done.

(a) In *recent traumatic cases* an attempt should always be made to restore the displaced viscera, after thorough purification, to their place in the peritoneal cavity. For this purpose the wound may be enlarged as far as necessary, and ribs resected sufficient to give free access. The rent in the diaphragm should at the same time be closed, and the opinion of authorities is unanimous that this can best be done from the thoracic side.

When symptoms of strangulation are present, other points arise for consideration. The principles which guide the surgeon under these circumstances are discussed in the next paragraph, *à propos* of strangulation of an established diaphragmatic hernia.

(b) Strangulation of an established diaphragmatic hernia is so seldom diagnosed that in nearly every case any operation which is performed will be in the form of an exploratory laparotomy. Even then it is by no means certain that the seat of constriction of the bowel will be detected, and numerous cases are on record where this has only been found after death. When the operator is fortunate enough to recognise the obstruction, however, authorities are agreed that the pleural cavity should at once be opened, by a U-, T-, or H-shaped incision, with resection of ribs, and the herniated bowel exposed before any attempt at reduction is made, for the following reasons:—(1) In this way the fatal error of drawing a piece of ruptured or gangrenous bowel into the peritoneal cavity in a situation so inaccessible as

the vault of the diaphragm will be avoided. (2) The condition of the prolapsed viscera can be determined, and steps taken to purify or repair it, as may be necessary, before its replacement. (3) It is found to be both easier and safer to reduce the hernia by pushing from above than by pulling from below. (4) The facility with which reduction is effected from above is doubtless due to the fact that the admission of air abolishes the negative pressure in the pleural cavity. (5) The thoracic opening permits of the purification and drainage of the soiled pleural cavity, and so diminishes the risks of empyema. (6) The closing of the opening in the diaphragm is only possible if attempted from the upper aspect.

While the opening of the pleura has these surgical advantages, it must at the same time be remembered that it adds to the already serious condition of the patient the risks incident to pneumothorax, and may turn the balance against him.

**INFLAMMATORY AFFECTIONS**—*Diaphragmatic pleurisy* often closely simulates the onset of acute perforative peritonitis and other surgical abdominal conditions.

An *abscess* may form in the substance of the diaphragm, as occurred in Meltzer's case, where a young child suffering from pneumonia developed symptoms simulating those of empyema. After exploratory puncture with negative result, the chest was opened, and a localised abscess found in the diaphragm.

*Perforations*, however, are much more common, the diaphragm becoming secondarily involved in inflammatory processes originating in neighbouring organs.

The primary seat of disease may be in the thorax, in the form of empyema, abscess, or gangrene of the lung, tuberculosis, suppurative pericarditis, or mediastinitis. After breaking through the diaphragm the inflammatory products may enter the peritoneal cavity, giving rise to a sub-phrenic abscess, or may pass between the peritoneum and the muscles of the back as a lumbar abscess, eventually opening to the skin. Where adhesions have formed between the diaphragm and some portion of the alimentary canal, a purulent collection, such as empyema, may discharge itself into the bowel, and be voided by the rectum.

On the other hand, the diaphragm may be perforated from below, a sub-phrenic abscess thus finding its way into the pleural cavity, lung, mediastinum, thoracic wall, pericardium, or even into the heart itself. The perforation usually takes place through one of the abnormally thin portions of the muscle, through one or other of the anatomical openings in the diaphragm, or at the seat of adhesions.

**SUB-PHRENIC ABSCESS** may originate in such a variety of conditions that it is by no means a rare affection. Owing to the frequency with

which such collections contain air the condition is sometimes referred to as a "sub-phrenic pyo-pneumo-thorax," a nomenclature which involves a contradiction in terms. Maydl's table indicates the different primary causes of sub-phrenic abscess, their relative frequency, and the proportion of cases in which there is air in the abscess cavity

Out of 179 cases collected by him, sub-phrenic abscess originated -

	Cases	Contained Air
In stomach and duodenum	35	20
" caecum and appendix	25	8
" liver or biliary passages	20	1
" internal injuries	18	3
" hydatid disease	17	3
" the intestines	13	4
As a metastasis	11	1
In inflammation round kidney	11	1
" miscellaneous conditions	11	5
" disease inside chest	9	1
" external injuries	6	0
" caries of ribs	3	0

These collections may be situated anywhere between the liver and the diaphragm, but are commonest in the left hypochondrium

Many of these primary conditions may perforate the diaphragm without first forming a sub-phrenic abscess. Thus Pick found that out of 28 cases of gastric ulcer leading to perforation of the diaphragm, 20 did so directly, while only 8 formed a sub-phrenic abscess. Ulcers of the fundus of the stomach, especially if associated with adhesions, tend to end thus more than others. Ulcers on the posterior part of the stomach by contracting adhesions obliterate the lesser sac of the peritoneum, so that when perforation takes place a sub-phrenic abscess results

In appendicitis the pus may reach the diaphragm on the inner side of, or behind, the peritoneum. The sub-phrenic abscess may develop very rapidly in acute suppurative cases, with perforation or gangrene of the appendix, especially when no adhesions have formed around the caecum, but as a rule it does not occur for some weeks. Unless such an abscess is evacuated, early perforation of the diaphragm is almost sure to follow. Thus out of 25 cases 11 were not operated upon, and all perforated. Of the 14 which were incised 9 were saved

**Clinical Features.**—The comparative frequency with which air occurs along with the pus renders sub-phrenic abscess very liable to be mistaken for pneumo-thorax or pyo-pneumo-thorax, while many of the cases in which there is no air closely simulate empyema

The onset may be acute, especially when due to perforation of a gastric ulcer, or there may be little or no evidence of the formation of a very large abscess

In addition to the general signs of pus forma-

tion, rapid or slow as the case may be, there is usually an excessive fullness and resistance in the region of the epigastrium or left hypochondrium, which often tends to point by the side of the ensiform cartilage

The liver dulness may be lost on account of the gas in the abscess cavity, and the liver is often markedly displaced downwards by the pus. The diaphragm, too, may be pushed up as far as the third or even the second rib. The heart also is often displaced

Clinical evidence of the existence of one or other of the conditions which give rise to sub-phrenic abscess, such as gastric ulcer, hepatic abscess, hydatid disease, empyema, etc., is of great diagnostic value

As distinguishing this condition from true pyo-pneumo-thorax, it is found that the hyper-resonant note is lower in the thorax, and extends into the upper abdominal regions, the pus lying still lower. Amphoric breathing and the bell-sound may be present

When a sub-phrenic abscess has perforated into the pleural cavity, the suddenness of the onset of thoracic symptoms, with a previously healthy condition of the chest, and the history of gastric, intestinal, liver, or other abdominal disease, render the diagnosis fairly clear

The prognosis is on the whole unfavourable. If left alone it usually proves fatal by setting up empyema, abscess of the lung, or peritonitis. Even when it opens into the alimentary canal a fatal issue is common

**Treatment.**—The only rational and efficient treatment is free incision and drainage

The causes, site, and site of these abscesses are so varied that no uniform method of operating is applicable to all. The physical signs, supplemented, if possible, by an exploratory puncture, will guide us to the correct situation for incision, which in all cases should be free, and should permit of thoroughly efficient drainage being established. The adhesions which form round the abscess usually shut off the general peritoneal cavity from infection

When the pus is on the convex surface of the liver, or on the upper aspect of the kidney or spleen, access may be got through the pleura, after resection of portions of two or more ribs, with the overlying muscle. The diaphragm is divided, after the pleural sac has been shut off, by a circular series of stitches. It has been urged against this method that the pressure of the large tube necessary for drainage may cause necrosis of the adjacent ribs.

Although the results, even after incision, are not uniformly successful, it has usually been found that failure has been due either to co-existent complications, such as peritonitis, pneumonia, or metastatic purulent collections, or to incomplete operation, such as neglect to drain a concurrent empyema, an outlying loculus of the abscess, or to inefficient drainage



**Diaphragmalgia or Diaphragmatalgia.**—Neuralgic pain in the diaphragm.

**Diaphragmatitis.**—Inflammation of the diaphragm

**Diaphragmatocele.** — Diaphragmatic hernia

**Diaphthora.**—Putrefaction, especially intra-uterine putrefaction following fatal death.

**Diaphysis.**—The body or shaft of a long bone, forming from the primary ossific centre. See PHYSIOLOGY, TISSUES (*Bone, Epiphysis and Diaphysis*)

## Diarrhœa.

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See AIR, EXAMINATION OF (*Ground Air*), ASTRINGENTS, BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Cerebral Anæmia, Causes*), CHILDREN, DEVELOPMENT OF (*Symptoms of Teething*), CHOLERA, EPIDEMIC, CHOLERA NOSTRAS, COLON, DISEASES OF, CONSTIPATION, ENEMATA, GASTRO-INTESTINAL DISORDERS OF INFANCY (*Diarrhœa, Simple, Cholera, etc.*), HYSSTERIA (*Disorders of Digestive Organs, Paroxysmal Diarrhœa*), INFANTILE DISEASES OF (*Enteritis, Ulcers, Lardaceous Disease, Malignant Disease*), LARDACEOUS DEGENERATION, LUNG, TUBERCULOSIS OF (*Complications, Alimentary, Diarrhœa*), MALINGERING (*Digestive System, Diarrhœa*), MEASLES (*Cow*), MENSTRUATION AND ITS DISORDERS (*Vicarious*), METEOROLOGY (*Seasonal Recurrence of Diarrhœal Affections*), MESENTERIC GLANDS (*Tuberculosis, Clinical Features*), MUSCLES, DISEASES OF (*Trichiniasis*), MYIASIS (*Intestinal*), NEPHRITIS (*Acute, Chronic*), PELLAGRA, PEMPFIGUS (*Acute, Malignant*), PERITONEUM, ACUTE PERITONITIS (*Symptoms, Dehydration, Diarrhœa*), PNEUMONIA, CLINICAL (*Childhood*), PHARMACOLOGY, PRESCRIBING, RECTUM, DISEASES OF (*Impaction of Fæces*), RHEUMATISM IN CHILDREN, SPIRUE (*Hill Diarrhœa*), TABES DORSALIS (*Intestinal Crises*), THYROID GLAND, MEDICINE (*Ætrophthalmic Gout, Digestive System*), TYPHOID FEVER (*Symptoms*), TRADES, DANGEROUS (*Lead-Poisoning*), URÆMIA (*Digestive System*), WATER (*Water-Poisoning, Diarrhœa*)

**DEFINITION.**—The term diarrhœa, which means literally "a running through," is applied to the frequent discharge of loose evacuations from the bowels. The too frequent passage of motions of normal consistence is not properly spoken of as diarrhœa. Diarrhœa may be due to increased peristalsis or increased intestinal secretion, or both. Although diarrhœa is, strictly speaking, a symptom only, there are many conditions

where it is practically of sole importance, in which therefore it may legitimately be regarded as the disease.

**ETIOLOGY.**—Amongst etiological factors common to different forms of diarrhœa *age* is of considerable importance, for although diarrhœa may occur at any age, it is most common and much more fatal at the extremes of life. Children under the age of two years are very liable to diarrhœa (to the extent of fully 80 per cent of fatal cases), and in old persons a severe diarrhœa often occurs as a terminal complication in chronic wasting diseases. The *temperature* of the atmosphere is also of importance. A sudden marked fall in the temperature is apt to be attended by a number of cases of diarrhœa, while during the hot months of the year children suffer severely, and the mortality from diarrhœal diseases varies almost exactly with the mean temperature. While diarrhœa has not been shown to vary with the density of the population, *overcrowding, want of cleanliness, and especially contamination of the food (milk)* supply must be regarded as important etiological factors.

**SYMPTOMS.**—The passage of loose alvine evacuations with the accompanying discomfort may be the sole symptom present. More commonly gastric disturbance, pain, flatulence, and other symptoms are present as well.

**Character of the Stools.**—The number of the evacuations varies greatly. There may be only two or three in the day, the patient feeling quite well in the intervals, or there may be four, five, or more every hour, and the feeling of discomfort and desire for a passage may be constant. The amount of the evacuations also varies within wide limits, from a comparatively trifling discharge to so copious a flux as to rapidly drain the tissues of the body. The motions may be quite liquid and even watery, or may be of the consistence of thick gruel. They may contain scybulous masses resulting from piecemeal constipation. Fluid motions are often described as resembling "pea-soup" (typhoid fever), "rice water" (cholera), "scrapings of meat" (dysentery), "frog-spawn" or "boiled sage" (dysentery with copious mucous discharge). In "hæmteric" diarrhœa food taken shortly before is passed unchanged. The colour of the motions varies according to the amount of bile present, and also with the nature of the food. Commonly they are light or dark brown or clay-coloured. They may be black or tarry from the presence of blood, or may be coloured by various drugs (bismuth, iron, hæmatovylum, etc.). The green colour so common in the diarrhœas of children may be due to biliverdin or to chromogenic bacteria (the green bacillus of le Sage). In some cases the evacuations have an extraordinarily offensive odour due to proteid decomposition. A cadaveric odour may develop

in association with necrosis of mucous membrane *Mucus* may be present in considerable abundance in catarrhal conditions of the large intestine, and the passage of mucous casts or "skins" is characteristic of a condition described as mucous disease or mucous colitis. *Fat* may be present in the stools in considerable abundance, sometimes largely in the form of fatty crystals, in association with deficiency of bile (so-called "acholic diarrhoea") or more characteristically of the pancreatic juice. *Pus*, gall-stones, enteroliths, intestinal parasites, fragments of gangrenous mucous membrane or of malignant tumours, may be discovered in the motions (see "Fæces").

*Pain*—Abdominal discomfort and even actual pain are present in most cases, usually most marked in the lower abdomen, and dependent on irregular peristalsis of the intestine and flatulence, parts of the intestine being spasmodically contracted, while neighbouring parts are over-distended by gas. The passage of the flatus is often attended by rumbling noises or borborygmi. Where there is much irritation in the rectum defecation is often attended by severe tenesmus.

*Fever*—In ordinary simple diarrhoea fever is either absent or slight and transient, but many of the diseases which cause diarrhoea are highly febrile.

*Constitutional*—When large liquid motions succeed each other rapidly as in choleraic diarrhoea the tissues are drained of fluid, the secretions, especially the saliva and the urine, are arrested, and the patient suffers severely from thirst, and soon sinks into a condition of collapse.

THE VARIETIES OF DIARRHOEA—Diarrhoea is a symptom of so many different pathological conditions that it is impossible to give any natural classification of the varieties met with. For convenience the following groups may be recognised—

(1) A large number of cases are due to *local irritation*, arising especially from the ingestion of improper food. Here we may also include diarrhoea due to excess of bile or to absence of bile, to intestinal concretions, to intestinal parasites, to purgative drugs, and to certain irritant poisons. A choleraic diarrhoea may result from arsenical poisoning. In these cases the irritation may lead to increased peristalsis, or to intestinal hyperemia and catarrh, or to slight or severe inflammation of the mucosa.

(2) Certain forms of diarrhoea have long been recognised as *eliminative*, especially the diarrhoea of uræmia. Diarrhoea considered to be of this character has been described as occurring in rheumatism, diabetes, gout, and in certain fevers, especially confluent smallpox, influenza, puerperal fever, and streptococcal infections. Diarrhoea due to exposure to cold is possibly sometimes of this nature. Watery diarrhoea

may be present during the disappearance of ascites or other effusions.

(3) *Nervous* (psychical) influences may give rise to diarrhoea, for example in persons about to undergo a surgical operation.

(4) Many instances of diarrhoea are *microbic* in origin. Under this heading are included those due to toxic substances arising from putrefactive processes in foods and beverages (meat poisoning, cheese poisoning, etc.). The specific organisms of typhoid fever, cholera, cholera nostras, certain forms of dysentery, tuberculosis, may be found in the stools. Several outbreaks of diarrhoea have been attributed to forms of the bacillus enteritidis (Gardner). The diagnosis of malarial diarrhoea has been made by the discovery of Laveran's plasmodium in red blood-corpuscles contained in the stools. Diarrhoea has been attributed, perhaps somewhat doubtfully, to certain Rhizopoda (monads) and Infusoria (*Cryptomonas intestinalis*, *Trichomonas intestinalis*, *Paramoecium coli*).

(5) Diarrhoea may be *secondary* and associated with inflammation or even ulceration of the mucosa of the small or large intestine. Such inflammation may be (a) catarrhal, arising in the course of the specific fevers, as a terminal process in wasting diseases, or as a result of portal obstruction, (b) croupous or diphtheritic, or (c) ulcerative, as in tubercle, cancer, dysentery, typhoid fever.

*Chronic Diarrhoea*—Chronic diarrhoea may follow an acute attack or may develop independently. It is usually associated with intestinal catarrh due to one of the conditions already mentioned. Intestinal catarrh associated with portal obstruction is not uncommon in elderly people, and is associated with diarrhoea, debility, and anæmia. Marked depression of spirits may also be present.

Mucous diarrhoea (mucous colitis) is a very chronic affection, but the severity of the symptoms varies greatly from time to time. It is characterised by the passage of the mucous casts spoken of above, and in some instances by severe enteralgia.

Tropical diarrhoea is a generic term applied to several forms of chronic diarrhoea which are apt to afflict Europeans who have been long resident in tropical climates.

*DIAGNOSIS*—Occasionally patients complain of diarrhoea who do not suffer from diarrhoea at all, but simply from some local irritation about the anus or rectum. When diarrhoea is present the important point in diagnosis is to ascertain its cause. If the attack is acute it may be possible to trace it to some indiscretion in diet, or to some special article of food which may have given rise to intestinal disturbance in all who partook of it, or to exposure to cold. If the diarrhoea is very severe, and attended by pain and vomiting, the possibility of its being due to

irritant poisoning, whether from putrefactive alkaloids or from inorganic poison, must be kept in view. Amongst articles of diet which have in many instances given rise to severe diarrhoea may be mentioned pork-pie, veal-pie, sausages, tinned meats and fruits, stale oysters, crabs, cheese, ice-cream.

In chronic diarrhoea a systematic examination of the evacuations, paying attention to the points mentioned in the paragraph on the "Character of the Stools," is of the first importance. Microscopic or bacteriological examination may lead to a correct diagnosis. Obviously the existence of any present or past disease which may be the cause of the diarrhoea must be ascertained. Digital examination of the rectum and examination with Kelly's rectal speculum will often give important information.

The diagnosis of the part of the bowel chiefly affected is usually difficult, and often impossible. In catarrh of the small intestine there are often severe colicky pains, and the stools are greyish-yellow or ochreous, not very frequent, and may contain partially digested food. There are no definite symptoms of duodenitis, but it is usually associated with gastritis, and often with jaundice. When the large intestine is affected, pain may be absent, or may be very intense. The evacuations have a soupy consistence, and may contain large quantities of mucus. Their passage may be attended by marked tenesmus if the lower part of the bowel is affected.

**TREATMENT.—Acute Diarrhoea.**—Acutedietetic diarrhoea requires little treatment in slight cases except rest and abstention from solid food, but in most cases recovery will be hastened by the administration of a purgative as soon as possible after the onset of the attack. For this purpose half an ounce of castor oil with 20 or 30 drops of laudanum is a favourite prescription. A dose of rhubarb is preferred by some. If the diarrhoea still continues twenty-four hours after the action of the purgative, an alkaline stomachic mixture with carminatives may be ordered, or moderate doses of aromatic chalk powder, chalk mixture, or bismuth. If vomiting is present as well as diarrhoea, ice may be given to suck, and small doses of bismuth and pepsin may be administered at intervals of a few hours will be found useful. Where small motions are being passed frequently, and accompanied by a good deal of irritation of the rectum, an enema of one or two ounces of starch with 20 to 30 drops of laudanum should be given.

Other forms of acute diarrhoea should be treated on similar lines. Rest in bed, warmth, and the restriction of the diet to milk or bland farinaceous food often make the patient comparatively comfortable even when the evacuations have previously been very numerous.

In nervous diarrhoea, which is usually recurrent, the patient should be taught to restrain the impulse as much as possible. If about to

be exposed to any condition likely to bring on an attack, a dose of bromide of potassium may be administered beforehand.

**Chronic and Secondary Diarrhoea.**—In all cases the diet must be attended to, and all articles of food which are not being properly digested must be forbidden. In some forms of tropical diarrhoea rest in bed and the absolute restriction of the diet to milk is found to be the most effective treatment. Prolonged rest in bed is often the best remedy for chronic diarrhoea in hysterical women.

A diet restricted more or less strictly to raw meat is found useful in some forms of chronic diarrhoea.

Where diarrhoea is associated with acidity of the intestinal contents, chalk or carbonate of bismuth may be ordered with some of the vegetable astringents, and with or without opium. In some cases, and especially when there are frequent watery discharges, the mineral astringents will succeed better, such as the permanganate or perchloride of iron, sulphate of copper, or nitrate of silver.

Antiseptics have been much used of late in the treatment of diarrhoea of microbial origin, and may be tried in any case where the odour of the evacuations is very offensive. Perhaps the most successful method of producing some approach to aëpsis in the bowel is to give one or two purgative doses of calomel followed by the continued administration of calomel in full doses. Such treatment, however, is obviously unsuitable for many cases of chronic diarrhoea, but calomel in repeated small doses, carbolic acid, biniodide of mercury, naphthaline,  $\beta$ -naphthol, salicylate and subgallate of bismuth are all useful.

Where the mischief is chiefly in the colon, daily irrigation with a large quantity of fluid should be carried out. For this purpose salt solution (one drachm to a pint) or a 1 per cent solution of boracic or salicylic acid may be used.

If ulceration of the colon is present, astringent injections are often of the greatest value, such as nitrate of silver in the proportion of a drachm to two pints of warm water.

**Diarrthrosis.**—A joint allowing motion in all directions, a free arthrosis.

**Diastopic Method.**—The method of diagnosing the nodules of lupus vulgaris by pressing a piece of glass on them, the accompanying hyperæmia can thus be made to disappear, but not the nodules (*Unna*). See SKIN, TUBERCULOSIS or (*Lupus, Clinical Features*).

**Diastase.**—A ferment existing in malt, which, acting upon broken starch granules, converts them into dextrin and maltose, in the process various dextrans (differing in their rotatory power, and in other particulars) are pro-

duced. See PHYSIOLOGY, FOOD AND DIGESTION (*Carbohydrates, Maltose*), TEMPERATURE (*Elevation from Injection of Diastase*)

**Diastasis.**—Separation of two contiguous bones or muscles (*e.g.* diastasis of the recti muscles of the abdomen, diastasis of the head of the humerus from the shaft, etc.) The word is derived from *Gr* *diastasis*, standing apart

**Diastemato-.**—In compound words *diastemato-* means referring to a congenital longitudinal fissure. Thus *diastematocheilia* is a longitudinal fissure in the middle line of the lip, *diastematomyelia* is a longitudinal median fissure of the urinary bladder, *diastematomyelia* is separation of the spinal cord into two lateral strands in the whole or (more often) in part of its extent, etc.

**Diastole.**—The relaxation-phase (*Gr* *διαστολή*, to expand) in the action of the heart. See PHYSIOLOGY, CIRCULATION (*Cardiac Cycle, Phases*)

**Diastrophe.**—A distortion or deformity

**Diathesis.** See also APPENDICITIS (*Etiology, Rheumatic Diathesis*), BRONCHITIS, BRONCHITIS (*Etiology, Diatheses*), CACHEXIA, CONSTITUTION, GOITRE, HEMOPHILIA, HYSTERIA, INSANITY, NATURE AND SYMPTOMS, LYMPHATIC SYSTEM, PHYSIOLOGY AND PATHOLOGY (*Lymphadenoma, Lymphogenic Diathesis*), TUBERCULOSIS, etc.—This term, derived from the Greek words *δια* and *τιθημι*, may be defined as the predisposition or constitution of the body, in virtue of which certain of its tissues or organs become, at one time or successively, the seat of affections similar in their nature, these affections presenting for their origin no other more potent or more definite cause. The nature and meaning of diathesis can probably best be explained by the following considerations

In order that a human being should live through the normal number of years, doing during this period the ordinary amount of work, and exposed during this period to the ordinary risks from cold or injury, it is necessary that each organ and tissue in that being should possess, from the beginning, a proper amount of what is called vitality. It is evident that if any one of these tissues or organs has been endowed at the beginning of life with less than its proper amount of this vitality, its store must become exhausted previous to that of the others. A more or less localised breakdown will then occur, as the result of which the whole being will suffer, or perhaps even the continuance of life will be rendered impossible. Just as the need of a fleet is dependent mainly on the speed of the slowest vessel in it, so the duration of life is dependent mainly on the store of vitality possessed by the weakest of the tissues or organs of the body

It is known that, as the result of differences in the surroundings and modes of life amongst individual human beings, the wear and tear of life is not always equally distributed over the different tissues and organs. In one individual, for example, the heart and blood-vessels are specially strained, in another the bronchi and lungs, in a third the kidneys, in a fourth the nervous system, and so on. It is known, further, that the amount of vitality with which the new being is endowed at conception is dependent on the vitality of the parents. Hence it follows that, although the offspring may be always potentially better than the parents, if the parents have lived under unhealthy conditions, if they have been weakened by disease or accident, or if they have been immature or too old, then the store of vitality transmitted to all or certain of the tissues or organs of the offspring will be deficient. Hence it will follow that, as the result of no special overstrain or no excessive exposure to cold or other morbid cause, a premature breakdown will occur. This, for obvious reasons, will tend to show itself specially in one or other of the tissues or organs, heart, lungs, nervous system, kidney, etc., and so, as the result of disease affecting one or other of those parts, the individual will be cut off long before the normal period of life is reached.

The precise nature of the disease changes which affect these parts is apt to vary. It may be an inflammatory change pure and simple, like a nephritis, it may be an inflammatory change due to the entrance of some organism, as a tuberculous phthisis or an endocarditis, it may be a so-called degeneration, as a fatty heart or an atheroma, or it may be a new growth, a cancer. In all cases, however, the great importance of the tissue vitality has to be borne in mind, and although it is always difficult, and oftentimes impossible, to say whether the surroundings or the constitutional condition has been the more important factor in causing a disease, there is no doubt that the capability of the physician to prognose and alleviate depends largely on his power of recognition and proper perception of the relationship between these two.

The duration of life being dependent on the power possessed by the tissues and organs of the body to maintain themselves against disease changes, and the diathesis being the predisposition to disease changes inherent in certain of these tissues or organs, it is evident that the number of diatheses should theoretically be very great. This number, indeed, should correspond not only with that of the tissues or organs, but also, at least to some extent, with the diseased conditions to which these tissues or organs are liable. Hence, as can readily be understood, a very great number of diatheses has been described. Among these the more prominent are the gouty, lithæmic, rheumatic, strumous, scrofu-

lous or tubercular, cancerous, nervous, inflammatory, gangrenous, scorbutic, hæmorrhagic.

But a moment's consideration will show that this number is by far too large. Many of these are simply modifications of the same diathesis, others are to be regarded as diseased states rather than constitutional predispositions or diatheses. In this article, therefore, detailed reference need only be made to a few, and by this selection an additional advantage is obtained in that only those diatheses are described which are more or less readily recognisable by objective appearances.

**Gouty**—In this diathesis the individual presents, at least until the middle periods of life, all the indications of robust vitality and of great bodily and mental activity. The frame is well built and well nourished, though tending perhaps somewhat to fatness. As might be expected, the digestive and assimilative powers are excellent, the teeth are good, the skin of the face is florid, over the trunk it is rather thick with active glandular organs, the hair is abundant, showing, however, rather early a tendency to turn grey. Owing to the excellent assimilative and digestive power, and the feeling of well-being and fitness which such a diathesis confers, gouty individuals are rather liable to excess in eating and drinking, and a tendency to a more or less plethoric condition supervenes, which in time gives rise not only to the special gouty changes in the fibrous tissues, but to the occurrence of allied morbid changes in the heart, arteries, notably the cerebral ones, urinary organs, etc. There is no doubt, however, that in its slighter degrees, should the tendency to excess be guarded against, this diathesis is one which is probably more than any other associated with long life and bodily and mental vigour. Insurance statistics seem to show that a family history of gout is often found amongst the best lives, and the saying of Sydenham that "more wise men than fools have suffered from gout" seems, if properly interpreted, to bear this out. A somewhat gouty habit is therefore not infrequently an advantage in life. Its disadvantages are indirect, being the result of its tendency to lead to excess, and consequently overstrain of the excretory organs.

**Rheumatic**—This diathesis is one which to some extent resembles the gouty. The individual is usually well built, well nourished, and muscular, having good digestive and assimilative powers. But the blood and circulatory organs seem to present a lack of nutritive vigour. The cheeks are often ruddy, but it is to be remembered that this may mean the reverse of the exuberant health which it might at first sight indicate. Professor Laycock used always to teach his students to be distrustful of a very healthy-looking complexion.

In rheumatic individuals the innate weakness in the blood often shows itself in early life by

supervention of anæmia, especially in females, the corresponding faulty condition of nutrition in the heart and blood-vessels by endocarditic changes. Moreover, as the result of comparatively trifling exposures, the characteristic inflammations of joints, serous surfaces, etc., are apt to arise.

**Scrofulous**—This diathesis, which comprises the strumous and the tubercular, is one in which probably all the tissues and organs of the body have been endowed with an abnormally low degree of vitality, but in which, owing to this cause, the absorbent surfaces and the glandular organs are specially prone to suffer from the entrance into them of the tubercle organism. Its objective characteristics can probably best be described by following the older writers, and considering it as divided into two classes—(a) scrofulous, and (b) tuberculous (Jenner), or as these have been aptly enough, though not quite correctly, termed, the ugly scrofula and the pretty scrofula.

(a) *Scrofulous*—Of this the following were the characteristics as given by the older writers—figure heavy, abdomen large, ends of the long bones rather large, shafts thick, skin thick and opaque, complexion dull and pasty-looking, face plain, lips and ale of nose thick, lymphatic glands very perceptible to touch, temperament phlegmatic, mind and body lethargic. Although certain distinct diseased conditions have been confused with the diathesis in this description, probably in rickets and possibly cretinism, the above yet forms a picture which is, to a considerable extent, true to life.

(b) *Tuberculous*—To this in its turn were ascribed the following characteristics—Figure slim, adipose tissue small in amount, ends of long bones small, shafts thin and rigid, limbs straight, skin thin and transparent, complexion clear, superficial veins distinct, blushing frequent, eyes bright, pupils large, eyelashes long, hair silken, face oval, good-looking. Children the subject of this diathesis are precocious, cut their teeth, run about, and talk early. In adults the nervous system is usually highly developed, and the mind and body are specially active, so that this diathesis is practically more or less identical with the nervous. It is not to be forgotten, further, that with it there is often associated a fair amount of muscularity and a considerable amount of physical strength. With it, as with the scrofulous, however, the constitution is faulty and the liability to the supervention of tuberculous diseases of all kinds is great.

Of the other diatheses the so-called cancerous, the scorbutic, and the hæmorrhagic require mention only. The first of these cannot readily be distinguished diathetically (see "cachexia"), the other two are in reality forms of disease.

**Diazobenzene**.—An artificial alkaloid, of a "double nitrogen type," having the formula

$C_6H_5N.N.OH$ ; it closely resembles tyrotoxin, the toxic ptomaine which has been isolated from poisonous cheese, milk, or cream. Diazobenzene sulphonic acid is used in testing urine (*vide infra*). See also TOXICOLOGY (Cheese).

**Diazo-Reaction.**—Ehrlich's diazo-reaction may be obtained as follows: a few ccs of urine are put in a test-tube, an equal quantity of a saturated solution of sulphamic acid in a solution of hydrochloric acid (50 cc to 1000 cc) is added, then also an equal quantity of a half per cent solution of sodium nitrite is added, the whole is then shaken, next 1 cc of ammonia is run slowly down the side of the tube and forms a colourless zone above the urine, and if the urine be normal a brownish ring is produced where the two fluids meet, but in certain cases a deep brownish-red ring forms (the diazo-reaction), the foam of the mixed urine and reagent is brownish yellow in normal urine, and rose-red when the reaction is present. The diazo-reaction is not a certain test for typhoid fever, for it is found also in malaria, tuberculosis, and typhus. See LUNG, TUBERCULOSIS (Complications, Uro-genital, Prognosis), MALARIA (Benign, Tertian Fever, Urine), TYPHUS FEVER (Period of Advance).

**Dibasic Acids and Salts.**—Acids (e.g. sulphuric acid) are dibasic when they contain (in each molecule) two atoms of hydrogen replaceable by a base, salts are said to be dibasic which are formed by the replacement of two such atoms by a base.

**Dibothriocephalus Vulgaris.** See PARASITES (Cestodes, Bothriocephalus latus).

**Dicephalus.**—United twins or double monster, in which the two heads and generally the two necks are separate. See TERATOLOGY (United Twins, Dicephalic).

**Dicheilus.**—The congenital anomaly known as "double lip", it is due to the presence of a fold of mucous membrane on the inner aspect of the upper or lower lip, giving to it the appearance of duplicity (Ballantyne, Antenatal Pathology, vol ii p 390).

**Dichirus.**—The rare teratological state known as fused or double hand, in which there are more digits than normal (7 or 8), but, as a rule, no thumbs, and in which there may be two ulnæ instead of a radius and ulna in the forearm (Ballantyne, Antenatal Pathology, vol ii p 586).

**Dichloroacetic Acid.**—Acetic acid in which two atoms of chlorine have taken the place of two atoms of hydrogen in the acid radicle, acetic acid is  $CH_3COOH$ , and dichloroacetic acid has the formula  $CHCl_2COOH$ , it usually occurs in the form of a very caustic liquid.

**Dichotomy.**—The division of the fee received by an operator or a consultant between himself and the general practitioner who has called him in, a reprehensible practice from the standpoint of professional ethics.

**Dichroism.**—The property of a substance by which it has one colour when viewed by transmitted light and another by direct light.

**Dichromatopsia.**—Colour-blindness in which only two of the primary colours can be distinguished, in contrast to monochromatopsia, in which the whole spectrum appears in shades of one colour (e.g. green).

**Dicrocoelium Lanceolatum.** See PARASITES (Helminths, Trematodes).

**Dicrotism.** See PULSE (Interpretation and Value of the Sphygmogram, Dicrotism and Hyperdicrotism), PHYSIOLOGY, CIRCULATION (Arterial Pulse, Pulse Wave).

**Dictyoid.**—Reticulated, from Gr δίκτυον, a net, and εἶδος, form, thus dictyopsia is the morbid state of vision in which net-like objects are seen before the eyes.

**Didactylism.**—The malformation of the hand in which two digits alone are found, didactylly, "lobster-claw deformity".

**Didelphys.**—The uterus didelphys is the most complete form of double uterus, there being two separate laterally-placed halves, each of which, however, has only one ovary and tube attached, the vagina may be single or double.

**Didot's Operation.** See DEFORMITIES (Hand and Fingers, Syndactylism, Treatment).

**Didymin.**—Testicular substance, recommended in sterility, neurasthenia, etc. See INDIGESTION (Treatment, Nervous Dyspepsia).

**Diencephalon.**—The inter-brain or thalamencephalon in embryology.

## Diet.

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PENDICITIS (*Treatment*), BERI-BERI (*Cause*), CONSTIPATION (*Treatment, Diet*), DIABETES MELLITUS (*Treatment, Dietetic*), GASTRO-INTESTINAL DISORDERS OF INFANCY (*Infantile Diarrhoea, Treatment*), GASTRO-INTESTINAL DISORDERS OF INFANCY (*Constipation, Treatment*), GOUT (*Dietetic Treatment*), HEART, MYOCARDIUM AND ENDOCARDIUM (*Treatment, Diet*), INDIGESTION, INFANT FEEDING, INVALID FEEDING, LIVER (*Constipation, Treatment*), LUNG, TUBERCULOSIS OF (*Therapeutic, Dietary*), NURSERY HYGIENE (*Diet*), OBESITY (*Treatment*), PHYSIOLOGY, FOOD AND DIGESTION (*Diet and Dietetics*), PREGNANCY, MANAGEMENT (*Diet*), PUERPERIUM, PHYSIOLOGY (*Diet*), PUERPERIUM, PATHOLOGY (*Septicæmia, Diet*), SCURVY, INFANTILE (*Etiology, Treatment*), SPURGE (*Treatment, Dietetic*), STOMACH AND DUODENUM, DISEASES OF (*General Etiology, Errors in Diet*), TYPHOID FEVER (*Treatment, Diet*), UNDULANT FEVER (*Treatment, Diet*)

A Food may be defined as anything which, when taken into the body, is able either (1) to build up or repair tissues, or (2) to supply material for the production of heat or muscular work.

A true food must therefore be either a tissue-builder or a source of potential energy. Dietetic substances which are unable to fulfil either of these functions may have important actions on the body, but cannot be regarded as foods in the true sense of the term. Such substances are tea, coffee, and the extractives of meat.

THE NUTRITIVE CONSTITUENTS of foods may be arranged thus —

Organic—

Nitrogenous	{ Proteids, e.g. myosin of meat, casein of milk
	{ Albuminoids, e.g. gelatine
Non-Nitrogenous	{ Carbohydrates, e.g. sugars and starch
	{ Fats, e.g. butter

Inorganic—

Water.  
Mineral matters, e.g. sodium, potassium, calcium and magnesium, iron, phosphorus, chlorine, sulphur.

The functions of food as building material, fuel, and a supplier of energy, are fulfilled by these different groups of nutritive constituents in varying measure.

*Building material* is supplied by the proteids, water, and mineral matters, and by these alone. Fats, carbohydrates, and albuminoids are unable to form tissue. Seeing that there is always a certain amount of waste of bodily structure going on there is a constant demand for these materials in the diet. Without them, or any one of them, life is impossible.

All the *organic* nutritive constituents of food serve as sources of heat. They are able to do so in virtue of their capability of undergoing oxidation in the tissues and organs. Fat requires the greatest amount of oxidation, and is therefore the most potent fuel food. Proteids, carbohydrates, and albuminoids are about equal as

heat producers, but are scarcely of half as much value as fats. The standard of heat production is the Calorie, which means the amount of heat required to raise the temperature of one kilo of water 1° Cent.

It has been found by experiment that—

1 gramme of proteid fully oxidised produces	4.1 Cal.
" " carbohydrate "	" 4.1 "
" " fat "	" 9.1 "

It is no doubt owing to their great value as heat producers that fats find such a large place in the dietary of the inhabitants of very cold climates, e.g. the Esquimaux. The presence of 40 per cent. of fat in the milk of the walrus is an instance of a similar adaptation on the part of Nature.

As raw material for the production of *muscular energy* the carbohydrates are now admitted to rank first. It seems certain, however, that the proteids can either directly or indirectly serve a similar function, and probably the fats are able to do so too. Whether the albuminoids are able to fill a similar rôle is uncertain. The great utility of sugar especially, as a muscle food, has recently been insisted upon by many observers, and its value is confirmed from such diverse sources as laboratory experiments with the ergograph, the efficiency of troops on the march, and the experience of Alpine climbers. It will probably win for itself a larger place than it has as yet taken in the dietary of training. Notwithstanding this outcome of modern research the necessity of a liberal supply of proteid in the diet of those who have to perform severe muscular work must not be lost sight of. Strenuous muscular exertion is always accompanied, sooner or later, by an increased excretion of urea. Whether one regards this as arising from the utilisation of proteid as a source of energy, or ascribes it simply to the destruction of muscle fibre which severe work necessarily entails, the loss still requires to be made good. That is the physiological justification for the high opinion which trainers have always entertained of a richly proteid diet for those engaged in athletic contests.

It is evident that the proteids are, by themselves, capable of fulfilling all the demands upon a complete food. They are able to build and repair the tissues of the body, to supply it with heat, and provide the muscles with pabulum for their work. It is this physiological omnipotence which gives to the proteids that pre-eminence which they possess amongst the nutritive constituents of food, and thanks to it, life can be maintained on a diet of lean meat and water, as in the Salisbury cure, for a practically indefinite time.

THE AMOUNT OF FOOD REQUIRED IN HEALTH

—An idea of the quantity of food required daily in order to maintain the body in a state of health can be arrived at either by a physiological or an empirical method. The former is

based upon estimations of the intake and output of the chief chemical constituents of the food, and of the amount of potential energy expended daily in the production of heat or muscular work. The latter is founded upon a simple analysis of the diets of healthy persons, living under known conditions, and neither increasing nor losing in weight. Diets based upon the results yielded by the first method are called *standard dietaries*, those which result from the second are *actual dietaries*.

(1) The chief chemical elements which the physiological method has to take into consideration are nitrogen and carbon. A healthy man, of average weight and doing a moderate amount of muscular work, excretes from 16-20 grammes of nitrogen and about 320 of carbon daily. In other words, he will require every day to be supplied with about  $\frac{2}{3}$  of an ounce of nitrogen, and  $\frac{3}{4}$  lb. of carbon, or one part of the former to every sixteen of the latter. The approximate proportion of carbon to nitrogen in some common articles of diet is as follows —

Roast Beef	has	1 of Nitrogen to 5 of Carbon
Eggs	have	" " 7 "
Cheese	has	" " 10 "
Milk	" "	" " 11 "
Pease	have	" " 12 "
Bread	has	" " 28 "
Potatoes	have	" " 33 "

Evidently none has nitrogen and carbon in proper proportion. For this reason one must either adopt a mixed diet or consume an undue amount of one or other element. The former alternative is the one usually adopted, and is illustrated by the following standard English diet (Olver) —

Foundation—	Carbon	Nitrogen
1 lb Bread	117	5.5
$\frac{1}{2}$ " Meat	31	7.5
$\frac{1}{4}$ " Fat	84	
Accessories—		
1 lb Potatoes	45	1.3
$\frac{1}{2}$ pint Milk	20	1.7
2 Eggs	15	2
2 oz Cheese	20	3
	335	21

Such a diet more than supplies the amount of carbon and nitrogen required.

Taking the output of heat and work as the basis of calculation, it is found that a man

doing moderate muscular work requires a supply of potential energy equal to 3000-3500 Calories daily. The next question is, what fraction of this total is to be provided in the form of protein, carbohydrate, and fat respectively? As regards protein the reply is easy. The amount must be sufficient to provide 20 grammes of nitrogen. Now one part of nitrogen is contained in  $6\frac{1}{2}$  of animal and 6.5-7 of vegetable protein. Hence  $20 \times 6.25$ , i.e. 125 grammes of protein, will be an ample allowance. The relative proportions of carbohydrate and fat are not so easily determined, for we have no means of knowing how much of the total CO<sub>2</sub> excreted daily is derived from the oxidation of the one and how much from the other. All we can do is to distribute the balance of Calories not obtained from proteins, between the two in accordance, as far as possible, with the results of actual observation of the usual amounts of each consumed by healthy men. As a matter of fact, the data given by different authorities on this point are conflicting, but 105 grammes of fat and 420 of carbohydrates may be regarded as a fair allowance, giving, if anything, in the direction of an undue preponderance of fat. These amounts will provide a total of 3211 Calories distributed as follows —

		Calories
Proteins	125 grammes $\times 11$	= 512.5
Fat	105 " $\times 9.3$	= 976.5
Carbohydrates	420 " $\times 4.1$	= 1722.0
		3211.0

Such a diet might be made up as follows (Atwater) —

	Oz	Calories
Beef	8	= 560
Fish (mackerel or herring)	4	= 230
2 Eggs		= 135
Butter	2 $\frac{1}{2}$	= 565
Cheese	1	= 130
Milk, 1 pint		= 325
Potatoes	8	= 180
Rice	2	= 205
Bread	9	= 720
Sugar	1 $\frac{1}{2}$	= 175
Total		3205

(2) A large number of *actual dietaries* have now been investigated, especially on the Continent and in America, the results of the more important of which are collected by Atwater in the following table —



## ACTUAL DIETARIES

Classes	Nutrients				Potential Energy of Nutrients
	Proteid	Fats	Carbo- hydrates	Total	
<i>European and Japanese dietaries</i>					
	Grams	Grams	Grams	Grams	Calories
1 Sewing girl, London, wages 93 cents (3s 9d) per week	53	33	316	402	1820
2 Factory girl, Leipzig, Germany, wages \$1 21 per week	52	51	301	406	1910
3 Weaver, England, time of scarcity	60	28	398	486	2138
4 Labourers, Lombardy, Italy, diet mostly vegetable	82	10	362	484	2192
5 Trappist monk, in cloister, very little exercise, vegetable diet	68	11	469	548	2304
6 Students, Japan	97	16	138	551	2343
7 University professor, Munich, Germany, very little exercise	100	100	240	440	2324
8 Lawyer, Munich	80	125	222	427	2401
9 Physician, Munich	131	95	327	553	2762
10 Painter, Leipzig, Germany	87	69	366	522	2500
11 Cabinetmaker, Leipzig, Germany	77	57	466	600	2757
12 "Fully fed" tailors, England	131	39	525	695	3053
13 "Well-paid" mechanic, Munich, Germany	151	51	179	681	2085
14 Carpenter, Munich, Germany	131	68	191	693	3194
15 "Hard worked" weaver, England	151	43	622	816	3569
16 Blacksmith, England	176	71	667	911	4117
17 Miners at very severe work, Germany	133	113	611	850	4195
18 Brick-makers (Italians at contract work), Munich	167	117	675	959	4611
19 Brewery labourer, Munich, very severe work, exceptional diet	223	113	909	1215	5692
20 German soldiers, peace footing	111	39	180	613	2798
21 German soldiers, war footing	131	58	489	681	3093
22 German soldiers, Franco-German War, extraordinary ration	157	225	331	773	4652

It will be observed that these correspond more or less closely, both in the proportions of their nutritive constituents and the total amount of potential energy yielded, with the physiological requirements already laid down and exemplified by the standard dietaries described above.

Roughly speaking, a man who adopts one of the above standard diets will consume about 3 lbs of solid food daily, containing about 23 oz of dry solids, or nearly an ounce per hour. The amount of mineral matter and water which he requires will be considered later.

It must be clearly realised that such standard diets are only of limited applicability. They cannot be rigidly enforced in all cases and under all circumstances. They are useful, however, as furnishing one with some idea of the amount and kind of nutriment which should be supplied to a healthy man doing a moderate amount of work. Thus they are of great assistance in constructing diet tables for public institutions in which the inmates are unable to have a free choice of foods, as, for example, in prisons, workhouses, hospitals, and the like. In applying them to individuals respect must be had to various modifying factors, of which the chief are age and sex, height and build, work and rest, climate and surroundings, and, last but by no means least, personal peculiarity. The influence of each of these may now be briefly considered.

**MODIFYING FACTORS**—(1) *Age and Sex*—Children require relatively more building material, fuel, and muscle food than adults. They require more building material because they must not only keep their tissues in repair but add to them by growth; they require more fuel because, like all small animals, they have a large surface compared to their bulk, and so lose heat rapidly; they require more muscle food because of their restlessness and bodily activity in play.

Hence children require a large amount of food, and the importance of proteid and fat to them especially can hardly be over-stated. With the aged the reverse holds good. The assimilative power of their tissues is on the wane and their bodily activities restricted, hence their diet should be smaller than that of middle life. One may easily underfeed the young, but one is apt to forget that it is almost as easy to overfeed the old. Women require relatively less food than men, not only on account of the lighter nature of their work, but also, probably, from their possessing a slower rate of metabolism. Roughly speaking, a woman may be regarded as requiring only 8 parts of food to every 10 required by a man.

As compared with the food of a man at moderate work, the proportions for children may be stated thus—

A child under	2 requires	0 ½
„ from 3 to 5	„	0 4
„ „ 6 to 9	„	0 5
„ „ 10 to 13	„	0 6
A girl „ 14 to 16	„	0 7
A boy „ 14 to 16	„	0 8

(2) *Height and Build*—These have a determining influence on the amount of food required by affecting (a) the body weight, (b) the extent of body surface. The greater the weight of the body the greater the amount of food required for its upkeep. Thus the heavy man requires more food than the light man.

The number of Calories which requires to be supplied per kilo of body weight are about as follows—

During rest	30 to 35
„ light work	35 to 40
„ moderate work	40 to 50

The greater the extent of body surface the more rapidly heat is lost and the larger the supply of fuel required. For this reason the tall thin man who has a large surface requires more food than the short stout man who has, relatively to his weight, a much smaller surface.

(3) *Work and Rest*—These two factors have a more potent influence on the amount of food required than any others. The necessity for a large supply of protein and carbohydrate during the performance of severe bodily labour has already been pointed out. The number of Calories required under such conditions may rise to 4000 or even 4500 (for examples, see Table I).

On the other hand, when the body is at complete rest, as is the case, for example, with an invalid lying in bed, the demand for potential energy may fall to 2000 Calories or less per day. Intellectual work does not appreciably increase bodily waste, and therefore does not demand a liberal diet. Quality, and especially easy digestibility, is a much more important consideration for the brain worker than mere quantity.

(4) *Climate* is probably of less real importance in determining the actual amount of food required than is commonly supposed. Fluctuations in external temperature should be met by diminishing heat loss through increase of the clothing rather than by increasing heat production by changes in the diet. Notwithstanding this, experience seems to show that in cold climates and in winter the fuel foods—and especially those rich in fat—should be increased, while in the reverse conditions the carbohydrates should be relatively increased, and the proteins and fats diminished. The blubber diet of the Esquimaux, on the one hand, and the rice diet of the Hindu on the other, are examples in point.

(5) *Personal peculiarities* as affecting the amount of food required are largely to be explained by the influence of body weight and

shape already considered. When all deductions are made, however, there is still room for the popular belief that some people can “get on” with less food than others, though both are living under the same conditions and performing the same work. Of this fact, if fact it be, there is no satisfactory explanation, but it is at least conceivable that some organisms are more economical machines than others, and turn their supplies of potential energy to better account, just as a man who has had practice will perform a given muscular feat with much less wear and tear than a novice. Such individual peculiarities may explain the tendency to obesity in certain families, and must always be borne in mind in regulating the diet of patients.

**THE RELATIVE VALUE OF FOODS**—The relative value of different foods must be decided on chemical, physiological, and economic grounds.

(1) The chemical value of a food is determined by the results of its percentage analysis. The composition of most of the foods in common use is set out in detail in the tables in the latter part of this article. It need only be remarked here that one cannot judge of a food from its chemical composition alone. A food which contains but a small percentage of nutrients can never be regarded as a valuable article of diet, but, on the other hand, the possession of high chemical qualities by no means necessarily implies suitability to the needs of the body.

(2) In judging of the value of foods on physiological grounds one has to consider (a) the digestibility of the food in the stomach, (b) its absorption in the intestine. A good food is one which is easily digested and well absorbed into the blood.

(a) The ease with which a food is digested in the stomach may be gauged by the time which elapses before it is passed on into the intestine. This time is occupied in bringing the food into a state of solution, and the more rapidly this is accomplished the greater is the digestibility of the food in question. As the results of experiments upon men the commoner foods may be arranged according to the length of time which they remain in the stomach, as follows—

1 to 2 hours	
7 oz.	water or plain tea, coffee, or cocoa
„	beer or light wine
„	boiled milk
„	herb tea
	Whites of 3 eggs
2 to 3 hours	
½ pint	water, beer, or boiled milk
A large	teaspoonful of coffee with cream, or cocoa and milk
2 raw	or poached eggs
½ oz.	raw meat
9 „	boiled calves' brains or sweetbread
2½ „	raw oysters
5 „	boiled white fish
7 „	cauliflower or asparagus.

5 oz	boiled or mashed potatoes
2½ "	white bread, old or new
2½ "	rusks
2 "	biscuits
3 to 4 hours	
8 oz	stewed chicken or roast partridge
9 "	boiled beef
½ "	beef steak
½ "	of any sort of bread or biscuits
½ "	of rice, spinach, apples, or carrots
4 to 5 hours	
4 oz	roast beef or steak
9 "	" goose
10 "	" duck
½ "	mashed lentils or stewed French beans
7 "	" peas

The articles in the first two groups are obviously those which make least demands upon the digestive powers of the stomach.

(b) The capability which a food has of absorption in the intestine must be clearly distinguished from its digestibility in the stomach. The most easily digested foods are by no means necessarily those which are most completely absorbed and *vice versa*. The following table (chiefly after Rubner) shows the degree to which the nutritive constituents of some typical foods are absorbed—

Food	Percentage Unabsorbed			
	Dry Substance	Protein	Fat	Carbohydrate
Rice	11	20.4		0.9
White Bread	12	21.8		1.1
Micronium	13	17.1		1.2
Boiled Beef	19	2.6		
Roast "	53	2.6		
Eggs	5.2	2.6	1.4	
Milk	8.8	7.1	5.3	
Cheese	6.4	3.3	5.2	
Meat	6.7	17.5		3.2
Peas	9.1	17.5		3.6
Whole Meal Bread	13.1	36.7		7.9
Potatoes (Mashed)	9.1	36.5		7.1
Cabbage	14.9	18.6		15.1
Carrots	20.7	39.0		18.2

It will be observed (1) that the carbohydrates and fats are much more completely absorbed than the proteids—indeed, it may be said that the fat of the diet is absorbed almost entirely, while the only foods in which there is any appreciable loss of carbohydrates are green vegetables and, to a less extent, the pulses, (2) that vegetable proteids are not nearly so well absorbed as those of animal origin. It must not be supposed that it is desirable that a food should be completely absorbed into the blood. The contrary is the case. The intestine demands a certain amount of insoluble residue or "ballast" to serve as a stimulus to its peristalsis. Hence the advisability of selecting foods which leave a considerable residue for cases of constipation, and of those which are very completely absorbed in cases of diarrhoea.

A study of the table will be of assistance in making such a selection.

(3) A good food must not only contain a high percentage of nutrients, and be easily digested and well absorbed, it must also be cheap. One may best arrive at the relative economic value of a food by calculating either the total number of Calories or the amount of building material obtained in it for a given sum. Thus one finds, proceeding on the first plan, that a shilling's worth of—

Wheat flour yields	13,782 Calories
Oatmeal "	9,189 "
Potatoes "	7,689 "
Beans "	7,630 "
Fat salt pork "	6,875 "
Sugar "	6,292 "
Margarine "	6,161 "
Cheese "	3,642 "
Butter "	1,082 "
Milk "	2,178 "
Leg of mutton "	1,076 "
Shoulder of beef "	870 "
Eggs "	768 "
Cod fish "	656 "

If the amount of proteid obtained be taken as the standard, one gets such results as these: 1 lb of proteid in peas costs 7d, oatmeal, 7½d, bread, 1s 6d, milk, 2s 2d, beef, 2s 8d.

A glance at the tables will show that both as regards the total number of Calories yielded and the amount of building material obtained, the vegetable foods are very superior to animal products. Amongst the cheapest of the latter in both respects are cheese and milk.

It must be clearly realised that the maxim "cheap and nasty" does not apply to foods. High price and high nutritive value are not synonymous. The price of a food in the market is determined more by such considerations as flavour and rarity than by chemical composition or digestibility. Thus a pound of cod at threepence yields just as much nourishment as a similar quantity of sole at eighteenpence, and common arrowroot at fourpence is of the same chemical and physiological value as Bermuda at two shillings.

#### ANIMAL FOODS

1 *Meat*—The flesh of animals, which constitutes meat, consists of muscle fibres held together by connective tissue. The fibres vary in length and thickness. If they are long and coarse, as in the leg of a crab, the meat is less easily digested than when the fibres are shorter and more delicate, as in the breast of a chicken. The fibres contain the muscle plasma, or "juice" of the meat, the chief ingredients of which are water, the proteid called myosin, a certain amount of hemoglobin, certain "extractive" bodies, the best known being creatin, and some mineral salts, chiefly compounds of potassium and phosphoric acid. Rigor mortis is due to clotting of the myosin, and meat in that condition is tough. By and by acids are developed

in the meat which soften the myosin, and the meat becomes tender again. Meat should therefore be eaten either before rigor mortis has supervened or after it has passed off.

The connective tissue of the meat consists of the substance called "collagen," which is converted into gelatine by boiling. The connective tissue is more abundant in old than in young animals, and the flesh of the former requires more cooking to convert the connective tissue into gelatine than that of the latter does. Embedded in the connective tissue is a certain amount of fat. In some kinds of meat, such as pork, the fat is abundant, in others, such as partridge and chicken, it is almost absent. In swimming birds, such as the duck and goose, too, the amount of fat between the fibres is relatively large. Fat so placed tends to hinder the access of the digestive juices to the fibres, and impairs the digestibility of the meat. It is well to forbid such meats to the dyspeptic. The chemical composition of the whole of the meat taken together is approximately this:—

	Water	Nitrogenous Matter	Fat	Mineral Matter
Lean	76.5	21	1.5	1
Medium	73.0	20.5	5.5	1
Fat	53.0	17	29.0	1

It will be observed that the fatter the meat the poorer it is in water. Fat and water are to a large extent mutually replaceable. Roughly speaking, three-fourths of ordinary meat consist of water. Of the nitrogenous matter, about 1 per cent consists of "extractives," and 2 to 3 per cent of albuminoids, such as collagen, the rest consists of proteid.

The objects of *cooking* meat are (1) to develop its flavour, (2) to improve its appearance by destroying its red colouring matter, (3) to sterilise it and so increase its keeping properties, and diminish the risk of infection by any disease germs or parasites which it may contain. The principles to be observed in carrying out these objects are—(1) to heat the meat to a temperature sufficient to sterilise it, and destroy its red colouring matter without over-coagulating its proteid, (2) to convert all the connective tissue as far as possible into gelatine, (3) to effect these changes without removing from the meat any of the "extractive" matter to which it owes its flavour. In carrying out the first principle, it should be borne in mind that the red colouring matter of meat is destroyed, and the proteid coagulated, at a temperature of 170° Fahr, and that to go above this simply tends to harden the meat by over-coagulation. In stewing as far as possible to convert all the connective tissue into gelatine, one must remember that

meat is a bad conductor, and therefore the heat should be applied to it slowly, and given plenty of time to act upon the collagen. The third object—the conservation of the flavouring ingredients of the meat—is to be achieved by "sealing up" the meat by a brief exposure of the surface to a temperature sufficient to suddenly coagulate all the superficial layers of proteid, and so prevent the subsequent escape of the extractives. The methods by which these principles are successfully carried out in practice are described in the article on "Invalid Cookery," but it may be pointed out here that they are best attained by heating the meat (after the preliminary sealing) for a long time to a temperature not exceeding 170° Fahr, and that the method of stewing or braising is more in accord with correct principles of cookery than any other.

The digestibility of meat is not improved by any method of cooking. Indeed, it may be laid down as a general rule, to which there are few exceptions, that cooking diminishes the digestibility of animal foods and increases that of vegetable products. The truth of this as regards meat is borne out by the following observations of Jensen:—

1½	of 1 raw beef are digested in 2 hours.
"	half boiled beef are digested in 2½ hours.
"	wholly boiled beef are digested in 3 hours.
"	half roasted beef are digested in 3 hours.
"	wholly roasted beef are digested in 4 hours.
"	raw mutton are digested in 2 hours.
"	raw veal are digested in 2½ hours.
"	raw pork are digested in 3 hours.

As has already been pointed out, meat is absorbed in the intestine very completely, only about 5 per cent of its dry matter being left behind.

The ease of digestion of raw meat and the smallness of the residue which it leaves in the intestine render it a food of special value in the treatment of certain affections of the stomach and bowels. It is best prepared by scraping the fibres away from connective tissue which holds them together by means of the back of a knife. The pulp so prepared may either be administered in the form of a sandwich, or, if that is inadmissible, it may be stirred into a small quantity of beef tea. Celery salt is a useful flavourer. Cakes of such beef-pulp, lightly browned on the surface, form the basis of the "Salsbury" cure.

The composition of some of the internal organs of animals used as food is as follows:—

	Water	Nitrogenous Matter	Fat
Kidney	76.7	16.9	4.8
Liver	69.8	21.6	5.4
Heart	62.6	16.0	20.4
Lung	79.7	16.1	3.2
Sweetbread	70.0	28.0	0.2
Tripe	71.6	16.4	8.5

Owing to their dense structure, kidney, liver, and heart are difficult of digestion. The lungs contain a large amount of indigestible elastic tissue. Blood is also sometimes used as food, but it contains only 18-23 per cent of solids, and is not well absorbed. The presence of much nucleo-proteid in all of the above organs, except the heart and tripe, makes it prudent for the gouty to avoid their use, seeing that nucleo-proteid is a source of uric acid.

*Jellies* are derived from gelatine produced by boiling the collagen of connective tissue. Young tissues, *e.g.* calves' feet, are the most abundant source. Isinglass is a pure form of gelatine derived from the swim-bladder of the sturgeon, but it is rather expensive, and does not really go farther than ordinary gelatine. Commercial gelatines (*e.g.* Nelson's or Cox's) are as good as any other for feeding the sick. The nutritive value of jelly is considerable. Six ounces (a large helping) of good jelly yields  $1\frac{1}{2}$  oz. of solids, of which about one-half is gelatine, the rest being sugar. This must be remembered in ordering jelly for diabetics. Gelatine being the best "protein-sparer," jelly is an admirable food for feeble patients.

The "extractives" of meat are the main constituent of *soups* and *beef extracts*. The physiological action of these extractives may be stated thus:—(1) They yield no potential energy, and are therefore not foods. (2) They have no power of increasing the rate or force of the heart. Then "stimulating" effect on the circulation is to be attributed to the hot water with which they are taken. (3) It is doubtful if they have any stimulating action on the nervous system, but they appear to remove the feeling of fatigue. (4) They taste and smell agreeably, and are, therefore, powerful aids to digestion by helping to call forth a secretion of gastric juice. (5) When taken in excess they are apt to excite diarrhoea.

Hence clear soups, which consist of a solution of the extractives, have no nutritive value, but are useful at the beginning of a meal. The same may be said of beef-tea as ordinarily prepared. The following is the latest analysis of Liebig's extract (Kemmerich).—Water, 18 per cent, proteins and gelatine, 30 per cent, extractives, 25 per cent, mineral matter, 20 per cent, other extract, 7 per cent.

It will be observed that it contains a considerable amount of soluble proteid, but as a teaspoonful of it only weighs 5 grains it can never be taken in sufficient quantity to be of real value as a food. The same may be said of those preparations, *e.g.* Bovril, to which some of the powdered fibre of meat has been added. The large amount of salts which beef extracts contain is of very doubtful advantage, for there is no proof that the demand for salts is increased in fevers, or that the mineral matter so supplied can be made any use of.

*Beef Juices* are preparations which contain the coagulable proteid of meat. The method of making raw beef juice is described elsewhere. It usually contains about 5 per cent of proteid. The amount in commercial beef juices varies from 20 per cent or more (*e.g.* Vitalin) to considerably less than 5 per cent (*e.g.* Valentine's), but the majority do not contain much more coagulable proteid than the home-made article. The large amount of extractives and mineral matter in the commercial juices is a positive disadvantage, as it renders it impossible to administer them in any considerable quantity, for that reason home-made juice is to be preferred. Beef juice is sometimes a useful article of diet in diarrhoea (especially in children), but it is difficult to administer anything like an adequate supply of nutriment in that form alone.

*Fish*.—There are two classes of fish—fat and lean. Lean fish is such as contains less than 2 per cent of fat (*e.g.* sole, flounder, whiting, haddock, cod, and trout). Of the fat fishes, mullet, halibut, and mackerel contain from 2.5 per cent of fat, the salmon, turbot, herring, and eel contain more than 5 per cent. The fat fishes are about equal in nutritive value to a similar weight of moderately fat beef, the lean fishes are poorer in nutrients than beef, and contain relatively more water and gelatine. All fish is poorer in "extractives" than meat, and for this reason fish is a less stimulating form of food than meat, and is sometimes preferable to the latter in some diseases, *e.g.* epilepsy. Lean fish is usually easily digested owing to the shortness of its fibres and the absence of fat, for this reason it is suited to invalids. Fat fish is difficult to digest, and the oil in it is apt to become rancid and irritate the stomach. It should be avoided by dyspeptics. The belief that fish contains much phosphorus, and is, therefore, peculiarly suited to brain workers, is entirely unfounded.

Allied to fish are lobsters, crabs, and oysters. These have the following composition:—

<i>Lobster and Crab</i>		
	<i>Fish</i>	<i>Body</i>
Nitrogenous matter	19.17	12.14
Fat	1.17	1.14
Water	76.6	84.31

<i>Oyster</i>		
Nitrogenous matter	.	6.2
Fat	.	1.2
Carbohydrate	.	3.7
Ash	.	2.0
Water	.	86.9

The coarseness of the muscle fibres in the flesh of the lobster and crab makes the limbs of these animals very difficult of digestion. Raw oysters are easily digested, but of low nutritive value owing to the high percentage of water. The nature of the carbohydrate material which they contain is doubtful.

**MILK AND ITS PRODUCTS**—Cow's milk has the following approximate composition:—

Water	87 to 88 per cent
Proteid	2 to 3 "
Fat	3½ to 4½ "
Sugar	4 to 5 "
Mineral matter	0.7 "

It fluctuates so greatly in composition, however, that one does not know within 30 per cent how much nourishment he is ordering in prescribing a given quantity of milk to a patient daily. Fat is the most variable element of all, and is usually taken as the criterion by which to judge milk. Good milk should contain 4 per cent.

The proteids of milk are caseinogen and lactalbumin, there being about one part of the former to seven of the latter in the milk of the cow. Caseinogen is a pseudo nuclealbumin. It yields no uric acid bodies, and no sugar on hydrolysis. This gives it a special value in gout and diabetes. Pure caseinogen is now prepared in the form of a flour (by the Protene Co.), and used in the preparation of diabetic bread and biscuits. "Nutrose" is caseinogen rendered soluble by being combined with soda.

The sugar of milk or lactose has the advantage of not being very sweet. It may thus be added to milk and other liquids, *e.g.* lemonade, to increase their nutritive value. Such addition is useful in the case of patients who are suffering from acute disease and are unable to swallow large quantities of nourishment. It is not fermented by yeasts, and may therefore be of no use in cases of dilated stomach, but is readily changed into lactic acid by certain micro-organisms, and in that form is a common cause of diarrhoea in infancy.

The chief mineral matter in milk is calcium, one litre of it containing about a gramme and a half of lime. Some of the calcium is combined with citric acid, the rest occurs as phosphate. Milk contains very little iron, four or five parts being required to yield the amount of iron (10 milligrammes) required daily.

The germs which are so apt to be contained in milk can most easily be killed by boiling. Pasteurisation—which consists in keeping the milk at a temperature of 70° Cent for twenty minutes—is sufficient to kill most disease germs, but does not destroy all spores nor the lactic-acid producing bacteria. It has the advantage of not affecting the taste of the milk. It may be carried out by placing the milk in stoppered bottles, and setting these in a deep saucupan of water heated to the necessary temperature.

The digestibility of milk is interfered with by the dense clot which it forms in the stomach. The density of the clot depends on (1) the amount of the casein, (2) the degree of acidity of the stomach contents, (3) the quantity of lime salts present. All of these factors have their

influence lessened by dilution. Water may be employed for the purpose, but barley-water or lime-water is better. The former acts mechanically by entangling the particles of casein. Lime-water seems to have a specific power of preventing clotting apart from its alkalinity. One part to two of milk is sufficient to ensure a loose clot.

A pint of raw milk remains about 3½ hours in the stomach, sour or skimmed milk about 3 hours, and boiled milk about 4 hours.

Milk is not very completely absorbed. When it constitutes the sole diet only about 90 per cent of its potential energy is really available. Thus it leaves a larger residue in the intestine than many other foods (see Table, p. 355). Children absorb it better than adults. Boiled milk is not *quite* so well absorbed as raw. The nutritive value of milk is high, but about 9 pints of it would be required daily by a man doing moderate work, 4 to 5 pints, however, are sufficient to maintain the nutrition of a sick person lying in bed. It contains too much water and too much proteid and fat in proportion to its carbohydrate, and is too expensive to justify one in regarding it as a perfect food.

*Cream* contains relatively much more fat than milk, but the actual amount varies greatly from 12 or 15 per cent in ordinary cream, up to 40 per cent or more in thick or separated cream. It contains as much sugar and proteid as a similar quantity of milk.

*Butter* has about 80 per cent of fat and a small amount of proteid, the remainder being water. Margarine, which is made from the more oily constituents of animal fat, has almost the same chemical composition, and is practically equal to butter in nutritive value.

*Koumiss* is produced from mare's milk by a combined lactic and vinous fermentation.

*Kepher* is a similar product prepared from the milk of the cow. In both the caseinogen is thrown down in a finely flocculent, and therefore easily digested, form, and is also partly peptonised, most of the sugar is converted into lactic acid, and a small amount of alcohol and a large proportion of carbonic acid gas produced. Three and a half quarts (a daily allowance) of koumiss yield 140 gms of proteid, 80 of fat, and 140 oz. sugar, with a combined fuel value of 1918 Calories. These preparations present milk in its most easily digested form, and owing to the destruction of much of the lactose, are better in some cases of diabetes than ordinary milk.

*Cheese* consists of the casein and fat of milk, modified by the growth in it of various micro-organisms, to the lypo-products produced by which the different varieties of cheese owe their distinctive flavour. The composition of the common cheeses is shown in the following table:—

COMPOSITION OF CHEESES<sup>1</sup>

Cheese	Water	Proteid	Fat	Ash	Average Cost per lb		Real Cost of 1 lb Nutrient	
					s	d	s	d
American	28.9	32.9	31	4.5	0	6	0	8½
Camembert	48.6	21.0	21.7	4.4	7d	each	1	1½
Cheddar	31.9	33.4	26.8	3.9	0	9½	1	2
Cheshire	34.2	29.4	30.7	4.3	0	9½	1	2
Cream	32.0	8.6	5.9	1.5				
Dutch	32.9	30.8	17.8	6.3	0	7	0	10½
Gloucester	31.9	36.7	24.7	4.4	0	9½	1	2
Gorgonzola	39.2	25.9	26.9	4.7	0	9	1	3
Gruyère	34.1	31.5	28.2	4.0	0	10	1	3
Parmesan	30.0	43.8	16.5	5.9	0	11½	1	1½
Rognonfort	25.1	34.8	31.5	5.5	1	1½	1	6
Stilton	27.6	23.9	38.9	3.1	1	2	1	7

<sup>1</sup> These figures are constructed by taking the averages of the analyses collected by Pearson and Moor. Prices are those of the stores.

Cheese is a condensed and cheap form of animal food of high nutritive value, and specially adapted to supplement deficiency of proteid in the diet. Its density and richness in fat, however, render it difficult of digestion. This difficulty is best overcome by dissolving the cheese and then mixing it with some cereal food, *e.g.* macaroni. Solution can easily be brought about by adding to every quarter pound of the grated cheese as much bicarbonate of potash as will lie on a threepenny piece, and stirring the whole with a little warm water or milk. The absorption of cheese in the intestine is fully equal to that of meat.

Eggs.—An ordinary hen's egg weighs about 2 ounces, of which about 12 per cent consists of shell, 58 per cent of white, and 30 per cent of yolk. The shell consists of carbonate of lime. The white and yolk have the following composition—

	Water	Proteid	Fat	Ash
White	85.7	12.6	0.25	0.59
Yolk	50.9	16.2	31.75	1.09

The white consists of a solution of various proteids, the chief of which is egg albumen. Some, at least, of these yield a reducing sugar on hydrolysis.

The yolk contains a large amount of emulsified fat and a considerable proportion of lecithin. It contains also other organic compounds of phosphorus, amongst them a nucleo-proteid and an organic compound of iron. Indeed, yolk of egg is one of the richest food sources of iron that we possess. The fact that it contains so much fat and organic phosphorus seems to make yolk of egg of peculiar use to growing animals, and explains its value in the diet of children.

Experiments on their digestibility have shown that two lightly-boiled eggs remain in the stomach about 1½ hours. If taken raw they remain rather longer, and raw eggs are not really more digestible than those which have been lightly cooked. Hard-boiled eggs remain about three hours. The absorption of eggs in the intestine

is very complete, only about 5 per cent of residue being left. Hence eggs may be safely ordered in diarrhoea.

The nutritive value of one egg is equal to that of about half a tumblerful of milk. The potential energy yielded amounts to 70 Calories. Twenty eggs are required to supply the amount of proteid required daily.

#### VEGETABLE FOODS

The vegetable foods are distinguished from animal foods mainly by containing much more carbohydrate and considerably less proteid. The carbohydrate is chiefly in the form of starch, although some vegetable foods, *e.g.* fruits, contain sugar instead. The proteids met with belong mostly to the globulin class, and contain less carbon and more nitrogen than animal proteids, which is said to give them a lower nutritive value. The comparative poverty of vegetable foods in proteid renders it difficult to obtain a sufficient supply of nitrogen from them alone unless a large quantity is eaten, and the consumption of vegetable foods in quantity is inconvenient owing to their bulk. This bulkiness is due to the fact that most vegetable foods contain much cellulose and much water, for even the dry vegetable foods, *e.g.* the cereals and pulses, take up much water in process of being cooked. Hence the pure vegetarian has to choose between living on a minimum of proteid or consuming such a bulk of food as throws a considerable strain on the stomach and bowels. The adoption of the former alternative seems to diminish one's energy, as distinct from muscular strength, and to lower one's power of resistance to disease, while the latter course is apt to culminate in disorders of digestion. The disadvantages of a purely vegetable diet are increased by the inability of the intestines to absorb large quantities of vegetable proteid, as already pointed out, unless presented in very special forms. On the other hand, their comparative poverty in nitrogen adapts the vegetable foods for use in chronic renal disease and in gout, while their bulkiness is of value in habitual constipation by stimulating peristalsis, and in obesity by affording a maximum of quantity with a minimum of nutritive material.

The vegetable foods may be divided into the following classes—

1 *Cereals*.—The composition of the most generally used of these is as follows.—

COMPOSITION OF CEREALS<sup>1</sup>

	Water	Proteid	Fat	Carbo- hydrates	Cellulose	Mineral Matter
Wheat	12.0	11.0	1.7	71.2	2.2	1.9
Oats	10.0	10.0	4.5	59.1	12.0	3.5
" (hulled)	6.9	13.0	5.1	66.6	1.8	2.1
Barley	12.3	10.1	1.9	69.5	3.5	2.4
Rye	11.0	10.2	2.3	72.3	2.1	2.1
Maize	12.5	9.7	5.4	65.0	2.0	1.5
Rice (unhulled)	10.5	6.8	1.0	68.1	4.0	4.0
" (hulk l)	12.0	7.2	2.0	76.8	1.0	1.0
" (polished)	12.4	6.9	0.4	79.1	0.4	0.5
Millet	12.9	10.1	2.9	68.3	2.9	2.2
Buckwheat	11.0	10.2	2.2	61.9	11.1	2.2

<sup>1</sup> The table represents the composition of the cereals in their crude form. The figures are compiled from a vast number of analyses, the data contained in the report on the composition of the cereals exhibited at the Columbian Exposition being freely used (U. S. Bull. 35). The proteid has been calculated from the nitrogen, using the factor 5.7 for all except barley, maize, and buckwheat, where the factor 6 was employed.

## COMPOSITION OF PRODUCTS DERIVED FROM CEREALS

	Water	Proteid	Fat	Carbo- hydrates	Cellulose	Mineral Matter
Wheat Meal	12.1	12.9	1.9	70.4	1.6	1.2
Fine Wheat Flour	11.0	11.5	0.8	75.3	0.7	0.7
Cracked	7.2	14.2	7.3	65.9	3.5	1.9
Roll'd Oats	7.2	15.4	7.2	61.8	3.5	1.9
Barley Meal	11.9	10.0	2.2	71.7	1.8	2.0
Pat'd Barley	12.7	7.4	1.2	76.7	0.8	1.2
Coarse Rye Flour	11.1	15.3	2.1	68.7	2.3	2.2
Finest "	11.2	6.7	0.9	80.0	0.8	0.1
Coarse "	11.1	8.7	4.6	72.3	1.1	1.3
" (fine)	12.5	6.8	1.1	75.0	0.8	0.9
Buckwheat Flour	14.0	7.1	1.2	73.9	0.6	1.2
Rye Flour (blended)	11.7	7.4	0.5	79.5	0.4	0.4

Of these wheat is mainly used in Europe, maize in America, and rice in the East. Maize is superior to wheat in fatty material, while rice is characterised by its small proportion of both proteid and fat. Oats are about equal to maize in fat, and superior to wheat in proteid, and rank as the most nutritive of all cereals. A plateful of porridge is equal in nutritive value to three slices of bread. The chief product of wheat is, of course, bread. The following is the average composition of white and whole meal bread respectively —

	White	Whole meal
Water	39	45
Proteid	6.5	6.3
Fat	1	1.2
Carbohydrates	51.5	45.3
Mineral matter	1	1.2

The greater moistness of whole meal bread causes it to be hardly at all superior to white bread chemically, while physiologically it is found to be not so well absorbed. It is therefore on no account to be preferred to white bread for ordinary use, though it is of value in cases of constipation. On the other hand, breads such as Hovis, which contain a large proportion of wheat germ, are peculiarly rich in proteid,

of which they may contain as much as 9 per cent.

Biscuits, being almost free from water, are more nourishing than bread, three pounds of the former being equal to five pounds of the latter. They are also very easily digested.

*Semolina* is prepared from the central parts of hard wheat. It contains 10.6 per cent proteid.

*Macaroni* and *vermicelli*, which are prepared from good wheat flour by mixing into dough and then drying, have a similar composition.

*Honny* consists of split maize and resembles the latter in its constituents, but has only  $\frac{1}{3}$  per cent of fat. *Cornflour* consists of practically pure starch prepared from maize.

2 The *Pulses* have the following composition —

## COMPOSITION OF PULSES

(From the Means of many Analyses)

	Water	Proteid	Carbo- hydrates	Fat	Cellu- lose	Mineral Matter
Green Peas	81.1	4.0	16.0	0.5	0.5	0.9
Beans	13.0	21.0	55.4	1.8	6.0	2.6
Lentils	11.5	24.2	58.4	2.0	2.0	2.7
House Beans (dry)	13.1	25.1	50.9	1.7	5.5	4.0
Broad or Windsor Beans (dry)	8.4	26.4	58.6	2.0	1.0	3.6
French Beans (Hans)	20.1	1.5	7.3	0.4	0.6	0.7
Peas (dry)	11.7	23.0	55.8	2.3	1.0	3.2
Haricots (H. Blancs) (cooked)	73.6	4.1	20.8	0.5	0.5	0.7
Soy Beans (stewed)	91.12	1.7	3.7	0.1	2.9	0.3
Soy Beans	11.0	32.9	25.7	18.1	4.4	1.9
Pea Flour	9.3	39.1	25.2	13.7	4.6	5.8
Pea Meal	8.3	21.0	17.0	41.1	1.1	1.9

They are richer in nitrogen than any other vegetable foods. Thus they owe to the presence of a vegetable proteid called legumin or vegetable casein. Legumin forms insoluble compounds with lime salts, which is the explanation of the advantage of cooking the pulses in soft water.

The pulses are somewhat difficult of digestion in the stomach, and are apt to produce a feeling of repletion, even in small amounts. In the intestine they are prone to excite flatulence owing to their richness in sulphur. Lentils have least tendency in this direction. As a group the pulses are well absorbed provided they be given in a state of fine division.

Soy beans and pea-nuts are largely used in the preparation of diabetical foods owing to their poverty in carbohydrates. The preparation Revalenta Arabica consists mainly of Egyptian lentil flour.

3 The roots and tubers consist chiefly of starch, as the following table shows —



## COMPOSITION OF ROOTS AND TUBERS

	Water			at	Fibre	Ash	Extrac- tives
Potatoes	76.7	1.2	19.1	0.1	0.6	0.9	1.4
" (boiled in skin)	73.8						
Carrots	8.5	0.5	10.1	0.3	1.5	0.9	1.0
" (cooked)	93.4	0.53	3.39	0.17	1.8	0.14	
Turnips	90.	0.9	5.0	0.15	1.8	0.8	1.1
" (cooked)	97.25	0.32	0.65	0.06	1.2	0.32	
Radishes	90.8	1.4	4.6	0.1		0.7	
Beetroots	83.9	0.5	11.0 <sup>1</sup>	0.1	3.0	0.9	1.0
" (cooked)	94.8	0.44	2.63	0.06	1.3	0.3	
Parsnips	80.1	1.1	14.1	1.0	2.1	1.3	
" (cooked)	97.28	0.22	1.16	0.29	0.72	0.12	
Artichokes	79.8	2.3	14.5	0.7	3.0	1.0	
" (cooked)	91.6	1.5	4.6	0.06	0.9	0.61	
Onions	89.1	1.6	6.3	0.3	2.0	0.6	
Sweet Potatoes	72.9	1.6	22.5	0.7	1.5	0.7	
Yams	79.6	2.2	15.3	0.5	0.5	1.1	

<sup>1</sup> 10 per cent of sugar

Of the small amount of nitrogenous material which they do contain a large proportion is in the form of amido bodies of little or no nutritive value. Hence they are too poor both in proteid and in fat to be fitted to constitute the main part of any diet.

It is worth remembering that potatoes contain considerably less starch than bread, and may consequently be allowed in larger proportion than the latter to cases of mild diabetes.

They owe their chief importance as regular articles of diet to the alkaline salts, especially of potash, which they contain. These are excreted in the form of carbonates in the urine, and for that reason green vegetables should be freely consumed by patients with a tendency to gravel. Their richness in cellulose makes them difficult of digestion, but useful as supplying "intestinal ballast" in cases of constipation. They contain so little carbohydrate that they

*Tapioca, sago, and arrowroot* are all preparations containing about 88 per cent of starch, and practically no nitrogen. Above they are of limited nutritive value, but are useful additions to foods, such as milk, which are richer in proteid. They rank amongst the most completely absorbed of all foods, which justifies their employment in cases of diarrhoea.

4. *Green vegetables* are of very low nutritive value, as is borne out by the following table representing their composition.

## COMPOSITION OF VEGETABLES

	Water	Nitro- genous Matter	Fat	Carbo- hydrates	Mineral Matter	Cellulose	Fuel Value per lb.
Caulage	89.6	1.8	0.4	5.8	1.3	1.1	Cals. 165
" (cooked)	97.4	0.6	0.1	0.4	0.13	1.3	
Cauliflower (head)	90.7	2.2	0.4	1.7	0.8	1.2	175
Sea kale	93.3	1.4		3.8	0.6	0.9	
" (cooked)	97.95	0.4	0.07	0.3	0.2	1.1	
Spinach	90.6	2.5	0.5	3.8	1.7	0.9	120
Vegetable Marrow	91.8	0.6	0.2	2.6	0.5	1.3	
" (cooked)	99.17	0.09	0.04	0.2	0.05	0.37	
Brussels Sprouts	93.7	1.5	0.1	3.4	1.3		95
Tomatoes	91.9	1.3	0.2	5.0	0.7	1.1	105
" (cooked)	94.07	1.0	0.2	0.1	0.76	1.5	
Greens	82.9	3.8	0.9	8.9	3.5		275
Lettuce	94.1	1.1	0.4	2.6	1.0	0.5	105
" (cooked)	97.2	0.5	0.16	0.5	0.1	0.9	
Leeks	91.8	1.2	0.5	5.8	0.7		150
Celery	93.4	1.4	0.1	3.3	0.9	0.9	85
" (cooked)	97.0	0.3	0.06	0.8	0.5	1.0	
Turnip Cabbage	87.1	2.6	0.2	7.1	1.5	1.3	145
Rhubarb	94.6	0.7	0.7	2.3	0.6	1.1	105
Macedoine (tinned)	93.1	1.4		4.5	1.0		110
Watercress	93.1	0.7	0.5	3.7	1.3	0.7	
Cucumber	95.9	0.8	0.1	2.1	0.4	0.5	70
" (cooked)	97.4	0.5	0.02	0.7	0.2	0.9	
Asparagus	91.7	2.2	0.2	2.9	0.9	2.1	110
Salsify (cooked)	87.2	1.2	0.08	9.0	0.3	2.2	
Endives	94.0	1.0		3.0	0.8	0.6	
Savoy	87.0	3.3	0.7	6.0	1.6	1.2	
Red Cabbage	90.0	1.8	0.19	5.8	0.7	1.2	
Sauerkraut	86.3	1.5	0.8	4.4	7.0		

<sup>1</sup> Probably only about a half of the nitrogenous matter consists of proteid.

may be used, with very few exceptions, even in severe cases of diabetes. Cauliflowers, being more easily digested than any other form of

They may be conveniently divided into "flavour fruits" and "food fruits." The former include all the berry class, and are chiefly eaten

## COMPOSITION OF FRUITS

	Water	Protein	Ether Extract	Carbo-hydrates	Ash	Cellulose	Acids
Apples	82.5	0.4	0.5	12.5	0.4	2.7	1.0
" (dried)	36.2	1.4	3.0	57.6	1.8		
Pears	83.9	0.4	0.6	11.5	0.4	3.1	0.1
Apricots	85.0	1.1	?	13.4	0.5		
Peaches	88.8	0.5	0.2	5.8	0.6	3.1	0.7
Greengages	80.8	0.4		14.4	0.3	4.1	
Plums	78.4	1.0	?	20.1	0.5	?	?
				(and Cellulose)			
Nectarines	82.9	0.6	?	15.9	0.6		
Cherries	81.0	0.8	0.8	10.0	0.6	3.8	
Gooseberries	86.0	0.4		8.9	0.5	2.7	1.5
Currants (red, black, and white)	85.2	0.4		7.9	0.5	4.6	1.1
Strawberries	89.1	1.0	0.5	6.3	0.7	2.2	
Whortlesberries (- Blackberries or Bilberries)	82.1	0.7	3.0	13.5	0.4		
Blackberries	88.9	0.9	2.1	7.5	0.6		
Raspberries	85.8	1.0	?	12.6	0.6		
Cranberries	88.5	0.5	0.7	10.1	0.2		
Grapes	79.0	1.0	1.0	16.0	0.5	2.5	
Melons	89.8	0.7	0.3	8.6	0.6		
Water Melons	92.9	0.3	0.1	6.5	0.2		
Bananas	71.0	1.5	0.7	22.9	0.9	0.2	
Oranges	86.7	0.9	0.6	9.7	0.6	1.5	small quantities
Lemons	89.3	1.0	0.9	8.3	0.5		
Pineapples	89.3	0.4	0.3	9.7	0.3		
Dates (dried)	20.8	4.4	2.1	65.7	1.5	5.5	
Figs (dried)	20.0	5.5	0.9	64.0	2.3	7.3	
" (fresh)	79.1	1.5		18.8	0.6		
Prunes (dried)	26.1	2.4	0.8	68.9	1.5		
" (fresh)	80.2	0.8	?	18.6	0.5		
Currants (dried)	27.9	1.2	3.0	65.7	2.2		
Raisins	14.0	2.5	4.7	74.7	4.1		

Composition of edible parts alone represented. Where cellulose is not given it is included with carbohydrates.

## COMPOSITION OF NUTS

	Water	Protein	Fat	Carbo-hydrates	Cellulose	Mineral Matter
Chestnuts (fresh)	38.5	6.6	5.0		45.2	1.7
" (dried)	5.8	10.1	10.0		71.4	2.7
Walnuts (fresh)	44.5	12.0	31.6	9.1	0.8	1.7
" (dried)	1.6	15.6	62.6	7.1	7.8	2.0
Filberts and Hazels (fresh)	48.0	8.0	28.5	11.5	2.5	1.5
" " (dried)	3.7	14.9	66.4	9.7	3.2	1.8
Sweet Almonds	6.0	24.0	51.0	10.0	3.0	3.0
Pistacio Kernels	7.4	21.7	51.1	14.0	2.5	3.3
Cocoa Nut (fleshy part)	16.6	5.2	35.9	8.4	2.9	1.0
" " (dried)	3.5	6.0	57.4		31.8	1.3
" " (milk)	90.3	0.5			9.0	

for the sake of their agreeable flavour. Like the green vegetables, however, they are also of service by reason of the vegetable salts of potash which they contain. The "food fruits," as represented by the fig, banana, date, and other dried fruits. They contain a large proportion of carbohydrate in the form of sugar, which gives them a quite considerable nutritive value, and

vegetable, may be advantageously recommended to the dyspeptic.

5. The *fruits* form a large class, whose composition is approximately as shown in above table

are used in many countries, *eg* Egypt, in place of the cereals. It should be remembered that the chief sugar met with in fruits is levulose, and that the latter is better utilised

by diabetics than any other form of carbohydrate

The *nuts* form a class by themselves, and are characterised by great richness in fat, as the preceding table shows. Their richness in fat and cellulose, and the compactness of their structure, render them difficult of digestion. Being comparatively poor in carbohydrates they are largely used in the manufacture of diabetic foods.

6 The *fungi*, *lichens*, and *algæ* are a small and unimportant group of vegetable foods. The first-mentioned are represented by the mushroom and truffle, which have the following composition—

	Mushroom	Truffle
Water	93.7	74.0
Proteid	2.2	6.1
Fat	0.3	0.6
Carbohydrate	1.2	10.2
Cellulose	1.1	6.4
Ash	0.1	2.0

These are very indigestible in the stomach—so much so, indeed, that the symptoms of indigestion which they are apt to produce have often been mistaken for poisoning. They are also very imperfectly absorbed, and cannot rank as foods of any value. The *algæ* are represented by Irish, and the *lichens* by Iceland moss. These have the following constituents (Church)—

	Irish Moss	Iceland Moss
Water	15.8	10.0
Proteid	9.1	8.7
Mucilage	75.4	70.0
Cellulose	2.2	3.5
Ash	14.2	1.5

The "mucilage" consists mainly of hemicellulose—a carbohydrate containing material of doubtful nature. It certainly yields a reducing sugar on hydrolysis. The nutritive value of these substances is very low, for a teaspoonful of good Irish moss jelly contains only about a shilling's weight of solid matter. Decoctions of them make pleasant demulcent drinks, but that is the limit of their usefulness in the sick-room.

**MINERAL CONSTITUENTS OF FOOD.**—About 5 per cent of the body weight consists of ash. Mineral matters are therefore to be regarded as tissue builders, and as such are essential to life, and as a matter of fact it is found that death ensues in a few weeks if no inorganic materials are supplied in the food. The chief mineral matters required are sodium and potassium, calcium and magnesium, iron, phosphorus, sulphur, chlorine, and traces of such substances as manganese and silica.

It is impossible to say exactly how much of any or all of these is required daily, for the reason, amongst others, that several of the mineral ingredients of the body are excreted by the large intestine, and one can, therefore, never distinguish those inorganic constituents of the feces which have simply not been absorbed

from those which have been absorbed and again excreted. An ordinary mixed diet, however, contains about 20 grammes of mineral matter, and that may safely be regarded as more than a sufficient supply. Further, it may be asserted that we know of no disease which is produced by an excess of mineral ingredients in the food, and of almost none, with the exception, perhaps, of scurvy, which is due to their deficiency.

*Potassium* is most abundantly represented in vegetable, and *sodium* in animal foods. The foods richest in *lime* are milk, eggs, cereals, and a few vegetables, such as radishes, asparagus, and spinach. Meat, fruits, and potatoes are poor in *lime*. It has, therefore, been recommended that one should forbid the former class and order the latter to patients who are suffering from atheroma. The utility of such a proceeding is, however, very doubtful.

About 10 milligrammes of *iron* are contained in an ordinary mixed diet (Stockman). The foods richest in that metal are yolk of egg, potatoes, bread, oatmeal, and rice. Milk is one of the poorest of all foods in iron, but five pints of it contain the requisite 10 milligrammes. The comparatively small amount of iron contained in ordinary food renders it impossible to look to dietetic means alone as affording a sufficient method of treating anæmias.

*Phosphorus* seems to be of special value in aiding the formation of new tissues. It is probable, however, that if it is to be available for that purpose it must be supplied in organic combination, and not simply as inorganic phosphates. The yolk of egg and tissues rich in nucleins, e.g. the roe of fish, sweetbreads, etc., are the richest sources of organic phosphorus. The importance of phosphorus as a brain food is probably overrated.

*Sulphur* is almost wholly taken into the body in the form of proteins, little or no inorganic sulphur being contained in an ordinary diet.

*Chlorine* is largely consumed in the form of common salt, of which about 10 grammes are usually added to the diet in addition to that which ordinary foods already contain. That the actual addition of salt to the diet is unnecessary is proved by the experience of those who live without it altogether. On the other hand, the evil effects attributed to its use are entirely imaginary. It is an undoubted aid to digestion, and, provided enough water be supplied as well, tends to limit rather than increase tissue waste.

#### BEVERAGES

The use of alcoholic beverages has been discussed under "Alcohol." We may therefore confine ourselves here to the consideration of water and the alkaloidal beverages (tea, coffee, and cocoa).

*Water* makes up about two-thirds of the

body. Like the mineral matters, it is thus an important tissue-builder and essential to life, death resulting in a few days if it be cut off. About 2½ pints of water should be consumed daily in addition to that already contained in ordinary solid food. Obviously, however, this quantity will vary greatly with such conditions as temperature and work. It is almost impossible, by the free drinking of water, to increase the amount of it in the blood, nor can one, by limiting the supply of water, render the blood appreciably poorer in it. On the other hand, it seems probable that the habitual ingestion of an excess of fluid may ultimately produce a more watery condition of the tissues, and if these be already water-logged a diminution of fluids in the diet may cause the passage of water from them back into the blood. This may explain the favourable results sometimes obtained by limiting the supply of fluids in cardiac dropsy.

Water is not absorbed in the stomach, but leaves it—if half a pint be taken—in the course of about half an hour. Even large quantities of water do not appreciably delay the digestion of food in the stomach, while in the intestine it probably aids somewhat the process of absorption. The rapid passage of water through the stomach makes it a peculiarly dangerous vehicle of disease. If the source of the water be at all suspicious, therefore, it is best to boil it, and re-aerate it in a gazogene, for no filter is really reliable for domestic use.

Water does not increase tissue waste, but merely washes out the waste products. For this reason its free use in gout, renal disease, and diabetes should be encouraged, and care should be taken to see that the patient gets enough of it in fevers, and if the pylorus is obstructed a due provision of water should be ensured by the use of saline enemata. Otherwise there is danger of the supervention of toxic symptoms from the retention of waste products.

*Aerated waters* are more favourable to digestion than ordinary water, for the carbonic acid gas which they contain acts as a stimulant to the stomach. This gas is rapidly absorbed through the stomach wall, hence it is well to avoid aerated waters in marked cyanosis. They should also be forbidden to patients in whom sudden distension of the stomach may be dangerous, e.g. cases of gastric ulcer or cardiac embarrassment.

**TEA, COFFEE, AND COCOA.**—*Tea* contains the alkaloid theine, along with some volatile oil, tannic acid, gummy matters, etc. Indian and Ceylon teas are richer in all the former ingredients than China teas, and green tea is richer than black. An ordinary teacupful of tea infused for five minutes will contain at least one grain of tannic acid and one or two grains of theine. The longer the tea is infused

—up to about half an hour—the more tannic acid will it contain. If one wishes to minimise the amount of tannic acid, one should use China tea and infuse it for four minutes only. The addition of milk also aids by precipitating the tannic acid.

*Coffee* contains caffeine (which is identical with theine), tannic acid, and an oily substance called cafeeol, besides other less important ingredients. A teacupful of black coffee has about the same amount of caffeine and tannic acid as a similar quantity of tea. "French" coffee consists usually to a large extent of roasted chicory, the chief ingredient of which is caramel.

*Cocoa* contains the alkaloid theobromine, but in small amount, it is closely allied to caffeine. In addition it has (in its ordinary dietetic form) about 26 per cent of fat, 12 per cent of proteid, and 25 per cent of so of carbohydrates, besides a considerable quantity of ash (about 4 per cent).

All of these beverages have a retarding influence upon digestion, the effect of tea being greatest. Coffee is apt to irritate the stomach by its cafeeol, it should therefore be forbidden in cases of gastric catarrh. Cocoa has least influence on the digestion of other foods, but the large amount of fat which it contains is apt to make it disagree.

The only member of the group which has any claims to be regarded as a food is cocoa. Seeing, however, that only about 10 grammes of cocoa are used to make a breakfast cupful of the beverage it cannot seriously aid in general nutrition. Indeed, seventy-five such cups would be required to supply the amount of potential energy required daily.

Tea and coffee are chiefly useful in virtue of the caffeine and volatile oil which they contain. These act as stimulants to the nervous system, and to some extent to the heart also, removing the sense of fatigue and increasing reflex action. The fact that they are usually taken hot no doubt contributes largely to their stimulating effects. The effects of tea and coffee on general metabolism is in the direction of increasing rather than of retarding body-waste.

These beverages are useful in nervous exhaustion and in alcoholic coma, they might also be used more largely than they are in fever. On the other hand, they should be avoided or used with great moderation by "nervous" people, and by those suffering from sleeplessness or palpitation. Their effects on digestion sometimes render it necessary to forbid their use in dyspepsia, although it is probable that the digestive disturbance attributed to the use of these beverages has been greatly exaggerated.

[Should the recent work by Chittenden (*Physiological Economy of Nutrition*, New York, 1904) be substantiated by other observers, our present conceptions of dietary standards will

need reconsideration. His observations, made on athletes, volunteers, and professional men, tend to show that health and efficiency are compatible with a diet containing much less than the accepted minimum of proteid. They extended over several months, during which considerable muscular work was done by the subjects without impairment of health or vigor, nor did the energy value of the diet require increase. Cluttennden's conclusion, that "the amount of proteid food needed daily for the actual physiological wants of the body is not more than one half that ordinarily consumed," has not as yet found general acquiescence, nor are independent materials for forming a judgment as to its correctness as yet available, but his results are so novel, and have attracted so much interest, that it is only right to draw attention to them here.]

**Dietary.**—An allowance or regulation of food, such as is set down as the rule in hospitals, workhouses, schools, or asylums, or a course of diet, such as is recommended in illnesses. See **Diet**.

**Dietetics.**—The science which deals with the regulation of the amount and character of food in health and disease. See **DIET**, **PHYSIOLOGY**, **FOOD AND DIGESTION** (*Dietetics*).

**Dlethyl.**—An isomeric form ( $C_4H_{10}$ ,  $C_2H_5$ ) of normal butane ( $C_4H_{10}$ ) it is found in petroleum.

**Diethylamine.**—Ammonia in which two of the hydrogen atoms have been replaced by ethyl radicals ( $C_2H_5$ ,  $C_2H_5$ ,  $HN$  or  $NH_2$ ).

**Diet's Crises.**—The sudden attacks of abdominal pain, accompanied by nausea, vomiting, and collapse, which occur in cases of floating kidney, and are probably due to strangulation of the kidney or to kinks in the renal vessels. See **CRISIS**.

**Dieulafoy's Aspirator.** See **ASPIRATOR**, **USES OF**.

**Diffusion.** See **PHYSIOLOGY**, **RESPIRATION** (*Interchange of Gases in the Lungs and in the Tissues*).

**Digallic Acid.**—Digallic or tannic acid ( $C_{14}H_{10}O_9 \cdot 2H_2O$ ) is an astringent acid found markedly in galls and also in the tissues of many plants. See **GALLS**, **TANNIC ACID**.

## Digestion and Metabolism.

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See also **ALCOHOL** (*Special Physiology, Digestion*), **BALNEOLOGY** (*General Balneo-Therapeutics, Digestive Disorders*), **URINATE** (*General Characteristics, Digestion*), **HEADACHE** (*Causes, Digestive Tract*), **KIDNEY, SURGICAL AFFECTIONS OF** (*Movable Kidney, Symptomatology, Gastro-Intestinal Symptoms*), **LEUCOCYTOSIS** (*During Digestion*), **PHYSIOLOGY, FOOD AND DIGESTION**, **STOMACH AND DUODENUM, DISEASES OF** (*Digestion in the Stomach and Duodenum*).

UNDER the vague term digestion all those processes may be included to which the food substances are subjected, and the resultant changes which the various food constituents undergo in order to prepare them for assimilation by the tissues. It therefore includes a manifold series of processes all tending towards the same point, namely, the transformation of substances, which in their original state could not be made use of by the cell, into pabulum which can easily be taken up, assimilated, and

used for the nutrition of the tissues. The most marked alterations which the food undergoes are brought about by secretions which special glands elaborate, the action being an extracellular one, and carried out mainly by the action of enzymes. These non-living ferments have specific actions on one or other of the food constituents. But there is an intracellular as well as an extracellular digestion, where the changes which the assimilated material undergoes can only take place in the living cell. Our knowledge of these intracellular changes is still very limited, and in the vast majority of cases we can at most say that certain constituents of the blood-plasma are taken up by a certain group of cells, and that they leave it in other forms of combination (see "Blood"). Most of our knowledge of such processes has been derived from the histologist, although the information so obtained is rather indefinite, owing to the great difficulties encountered in comparing structural with chemical alterations. It is almost impossible to know definitely whether there be at the root of all cellular digestion (e.g. auto-digestion of organs) the action of enzymes, and it is also difficult to be certain whether the ferment has been formed in the cells of a particular organ, or transmitted to the latter as a zymogen from other parts. The decomposition of the food principles into smaller molecules takes place gradually, the final products of metabolism being formed in the tissue cells where these bodies, which are circulating in the blood, and are required for the special work of the cell, are selected, and are in part used up to furnish energy for its specific life processes, in part also stored up either for its own use or that of other cells. The final transformations result always in the production of work and heat, and those products which cannot be used further by the organism are excreted, then further decomposition taking place outside the animal body. One of the most marvellous things in the cell life is the peculiar stamp which each cell or group of cells gives to its work, although the ultimate principles, whose decomposition affords the energy necessary for the particular rôle of the tissue, are the same. What is ordinarily understood by the term digestion, however, is the series of changes which the food undergoes in the alimentary canal, because it is there that the most marked alterations take place, and it is with pathological disturbances from this side that the physician has to deal in cases of disordered digestion. The food is subjected in the alimentary canal to processes of two great types—mechanical and chemical. They are not, however, independent of one another, and both are under the influence of the nervous system. In order to prepare the food for absorption, it is necessary to break it down and transform it into material which can pass into the blood or lymph.

### SALIVARY DIGESTION

*Salivary digestion* has two functions to perform in the first place, the preliminary preparation of the food-stuffs for digestion with the saliva, gastric, pancreatic secretions, etc., and, secondly, a specific action, the transformation of insoluble carbohydrates into dextrins and a soluble sugar, maltose.

The saliva is formed from the secretions of the parotid, submaxillary, and sublingual glands chiefly, with the addition of a mucous fluid from the buccal glands. It always contains suspended material—for example, epithelial squames from the palate, salivary corpuscles, leucocytes from the tonsils, etc., but when freshly secreted the fluid is clear, rapidly becoming cloudy, however, owing to a precipitation of calcium carbonate from the removal of the carbonic acid which kept it in solution. Normally it reacts slightly alkaline, but it may be neutral or slightly acid from lactic acid fermentation set up by organisms acting on food remnants which have adhered to the teeth. Its stringy like consistence is due to admixture with mucus. The specific gravity varies from 1.002-1.008. The principal constituents are water, salts (especially chlorides and phosphates of sodium, sulphocyanates), mucin, a diastatic ferment termed ptyalin, and a trace of albumin. Oxygen, carbonic acid, and a fairly large quantity of nitrogen are present. The most important constituent is the *enzyme*, *ptyalin*, which is produced by the serous salivary glands, although it is not present in the cells in the active form, but rather as a forerunner or zymogen. The activity of this enzyme is destroyed by warming the saliva to a temperature above 70° C. One may say that it is most active in a slightly acid medium (due to organic acids) at a temperature between 35° and 37° C. The action takes place rapidly, and is best studied by mixing saliva with muciilage of starch, and keeping the mixture at the above-mentioned temperature. The fluid first becomes clearer, and then the action runs through the later stages so rapidly that on examination, even after a few minutes have elapsed, the final products of the digestion have been formed. The first body that is formed is soluble starch or amylum, which takes up water, and is split up into a form of dextrin—erythrodestrin—and maltose. From the former another dextrin—achroodestrin—and maltose are formed. Erythrodestrin forms a coloured compound with iodine (of a red tint), while achroodestrin does not. In addition to maltose there is probably formed a small quantity of another sugar, isomaltose, and perhaps glucose. Sulphocyanates are by no means always present in saliva, only their presence or absence has no acknowledged pathological significance.

The most important *pathological constituents* are lactic acid in diabetes (never glucose), uric

acid in gout, and urea in various nephritic conditions. Certain drugs may be excreted by way of the saliva, *e.g.* mercury, iodine, bromine, etc. Blood or pus may be present in inflammatory conditions of the mouth.

*Nervous Mechanism of Secretion*—As a rule no saliva passes out from a salivary fistula unless there be chemical or mechanical stimulation of the surrounding parts, but the mucous secretion seems to be constantly produced. In man, however, even the sight or smell of food may set up salivation. If the glands be active, stimulation of the special taste nerves, or the sensory nerves of the mouth, will at once produce a flow of saliva. Mastication greatly increases the flow, owing to a more frequent stimulation of the nerve endings in the buccal mucous membrane by the substances which are undergoing solution or maceration. Severe irritation of the stomach, as in emesis, will produce salivation by means of the stimulation of the vagus. There are two great sets of nerve fibres passing to the salivary glands, cranial and sympathetic, both containing secretory fibres, and in addition the former contains vaso-dilator, and the latter vaso-constrictor fibres. The same nerves pass to the submaxillary and sublingual glands, while the parotid has a special supply. It is unnecessary to describe in this place the course of these fibres, suffice it to say that the cranial supply of the two first-mentioned glands is the chorda tympani branch of the facial passing through the lingual branch of the fifth nerve, while that of the parotid is from the glossopharyngeal, through Jacobson's nerve, the small superficial petrosal, the otic ganglion, and the auriculo-temporal branch of the fifth nerve. All three glands have a supply from the cervical sympathetic. On stimulation of the cranial branches there is an abundant watery secretion, poor in solids, while after stimulating the sympathetic, a sparse secretion, rich in solids and of stringy-like consistence, is produced. Reflex stimulation from the mouth under ordinary conditions affects simply the cranial nerves, and interruption in this reflex may occur, so that stimulation of the gustatory nerves will not produce an effect. During secretion the temperature of the saliva rises slightly above that of the blood in the carotid, and its pressure may also rise above that of the carotid. During active secretion an electrical current is produced in the gland, "the current of action." Nicotin and atropin paralyse secretion, the former affecting probably the nerve terminations around peripheral ganglia, the latter the final terminations around the secretory acini.

*The morphological changes in the salivary glands during secretion are of importance. It is customary to speak of the parotid as a true serous gland, the sublingual as a mucous, and the submaxillary as a mixed gland, but the*

human sublingual gland in all probability produces a mixed secretion, because the crescentic cells lying at the periphery of the mucous acini are serous in type. Serous cells, as, for example, those of the parotid, contain at the beginning of salivary digestion numerous granules in the zone next the lumen of the acinus, rendering the cell cloudy in appearance while the peripheral part around the nucleus is clearer. Gradually these albuminous granules are discharged, and the whole cell appears less opaque. The cells which secrete mucin appear clear and distended before discharge of their contents, while after secretion they are collapsed. Slowly mucinogen granules form for a little distance around the nucleus, and then, on taking up water, these swell and distend the free part of the cell. In all, the type of secretion is the same, material being taken up and stored in the outer part of the cells, and then gradually transformed into mucin or albuminous material in the inner part and passed out as the true secretion of the acini. The part which the nucleus plays in the process is still doubtful.

*The amount of saliva secreted during the day varies on an average about  $\frac{1}{2}$  to 2 litres. There may be a marked increase, as, for example, after mercury, potassium iodide, pilocarpine, and many vegetable irritants which stimulate the buccal mucous membrane. It may also be increased in many cases of nervous shock, while it is often greatly decreased in febrile conditions, and may be absent altogether in hysteria. Pathological changes in the salivary glands will be referred to under their special headings.*

#### GASTRIC DIGESTION

*Gastric Digestion*—Gastric digestion is in part a mechanical process, the food substances being brought into a condition of finer subdivision by means of the movements of the stomach, in part a chemical one, the finely subdivided food substances being gradually converted into a more soluble form, and one therefore better fitted for absorption. Under normal conditions the process of digestion is unaccompanied by sensation.

It is perhaps best to consider first the chemical process of digestion, prefacing this with a short account of the *chemical composition of the gastric juice*.

Gastric juice, as obtained from a case of gastric fistula in the human subject by Schmidt, was found to be a colorless transparent fluid of a specific gravity of 1.0022-1.0024. On boiling it becomes faintly opalescent, but yields no precipitate. Under normal conditions it appears not to be formed except as the result of stimulation, but even in a state of hunger the stomach is rarely found quite empty. The quantity present varies considerably. Anything above 50 cc may be regarded as pathological. Many observers hold that the small

quantity of 10-20 cc. normally found during hunger has been secreted as the result partly of stimulation due to the swallowing of saliva, partly of the mechanical stimulation arising from the introduction of the gastric food. The degree of acidity varies considerably even in the same individual, but 2 per cent may be regarded as a normal average.

In the dog, from which gastric juice can be obtained in considerable quantities, and free from the constituents of the saliva, and in which its composition has been much more carefully studied than in man, it is a colourless, odourless, transparent, strongly acid (containing about 5 per cent hydrochloric acid), and actively peptic fluid of an average specific gravity of 1.0047. It may be kept an indefinite time without showing signs of putrefactive decomposition. It is slightly dextro-rotatory, and gives the usual protein reactions. On being cooled to 0° C. a fine precipitate of great peptic activity forms. The chief organic constituents are the enzymes, pepsin, rennin, and possibly invertin, along with traces of mucin and proteid convertible into albumose and peptone on standing. The inorganic constituents are chlorides of sodium, potassium, calcium, as well as traces of magnesium and iron. Phosphates are also present in very small quantity.

The free acid of pure gastric juice is hydrochloric, the most important proof of this being that the quantity of chlorine present is greater than the chemical equivalent of the total inorganic bases.

*Conditions influencing the Secretion Sources and Mode of Formation of the Constituents of the Gastric Juice.*—The secretion is normally intermittent. It may be induced (1) by psychical stimuli, e.g. the sight, or even the thought of food, (2) reflexly by stimulation of the nerves of taste, even where no food is allowed to enter the stomach, as in the pseudo-feeding of dogs after the establishment of a Pawlov fistula, and, lastly, by the entrance of food or other substances into the stomach.

Simple mechanical stimulation of the gastric mucosa produces only a slight local secretion. It is unlikely, therefore, that the normal secretion is at all largely dependent on mechanical irritation. Digestible solid substances mixed with saliva on their entrance into the stomach induce an immediate slight local secretion due to mechanical irritation. After about fifteen minutes there follows a second secretion of gastric juice in much larger quantity from the whole surface of the gastric mucosa. The latter secretion is believed to follow as a result of the absorption of the small quantities of albumose and peptone produced by the action of the gastric juice secreted in consequence of the mechanical stimulation.

In the earlier stages of digestion the acid of the gastric juice is neutralised by the alkali of

the food and of the saliva. For a variable period (three-quarters of an hour according to van der Velden) free hydrochloric acid cannot be detected, the hydrochloric acid entering into loose combination with the albumoses, peptones, and albumins (to form acid albumin in the latter case) as quickly as it is secreted. During this period—the amylolytic stage of gastric digestion—the ptyalin of the saliva continues to exert its action upon the carbohydrate material of the food. As digestion proceeds the acidity of the juice gradually increases.

The hydrochloric acid and pepsin of the gastric juice are produced by different cells in the gastric glands. In the cardiac region of the stomach two varieties of cells are found, the centrally placed, chief or adenomorphous cells, and the parietal, adenomorphous or oxyntic cells. In the pyloric region one kind of cell alone is present, and that resembles the central cells of the cardiac region very much more closely than the parietal ones. The pyloric secretion, when obtained free from the cardiac one, is found to be alkaline, and when acidified with hydrochloric acid it possesses peptonising power. The amount of pepsin which it contains is, however, much smaller than that of the cardiac region. The secretion of the cardiac region, on the other hand, contains both pepsin and hydrochloric acid. These, along with other facts, indicate the parietal cells to be the producers of the hydrochloric acid, and the central cells to be the originators of the pepsin. The site of the formation of the rennin ferment or its precursor is uncertain. With regard to the histological appearance of the gland cells only a few words are necessary. The central cells in the resting condition are filled with distinct granules, which, during digestion, decrease in number. Typically, the granules are situated chiefly in the inner or luminal parts of the cells, the outer zone of the cells showing a complete absence of granules. In other later cases the granules are found scattered throughout the whole body of the cell, and the decrease is a general one.

In the ovoid parietal cells the granules are much smaller, and the changes not so distinct.

The granules in the central cells consist not only of fully formed pepsin, but of a precursor or zymogen, which, under the influence of dilute acids, and also during active secretion by the cell, is converted into pepsin. To give a full proof of this statement would take up too much space. The salient points of difference between the zymogen (pepsinogen) and the enzyme, pepsin, are that the former is insoluble in glycerine, not easily destroyed by alkaline solutions, and possesses no digestive activity until converted into pepsin by dilute acids. The latter, on the other hand, is soluble in glycerine, very easily destroyed by dilute alkalis, and is an active digestive enzyme.



The distinction of rennin from its zymogen is founded on similar facts

*The Nervous Mechanism of Secretion* - The secretory nerves are the vagi. In a dog, in which a gastric fistula had been established and the œsophagus cut through, the eating of food induced an immediate secretion of gastric juice, accompanied by reddening of the gastric mucosa owing to vascular dilatation. This reflex secretion ceased to occur after both vagi had been divided. Stimulation of the peripheral ends of the vagi\* slowly repeated induction shocks also produced secretion.

In a human subject in whom the lumen of the œsophagus had become closed so that a gastric fistula had to be formed, the chewing of food, none of which entered the stomach, produced a copious flow of gastric juice.

*The Motor Mechanism of Gastric Digestion* - The stomach is usually closed at both its orifices, the cardiac and pyloric openings, by the contraction of the sphincter-like, circularly arranged muscle fibres. The cardiac orifice is closed during each act of swallowing at the close of the peristaltic contractions of the œsophagus, and the pylorus is open from time to time to allow the passage of the liquefied food material or chyme into the intestine. During the process of digestion the stomach walls are in continual peristaltic movement, thus ensuring the thorough admixture of the food with the gastric secretion, and also permitting the mechanical subdivision of the food masses. By these contractions the food material is propelled in two directions, the circumferential portions of the food passing towards the pylorus, while the central portions pass towards the cardiac orifice. This circulatory movement brings the digestive secretion into intimate contact with the food substances. The muscular movements are most active in the region bordering upon the pylorus (antrum pylori), which for this reason is sometimes separated by an oblique furrow from the remainder of the stomach. In the early stages of digestion the pylorus is firmly closed, but towards its termination the closure becomes much less firm, so that the contractions of the antrum pylori succeed in propelling the more fluid portions of the mass through the pyloric opening. Cold water is said by some to produce rapid opening of the pylorus.

The gastric movements are brought under the control of the nervous system by means of two sets of fibres. The vagi contain accelerator fibres, so that their stimulation induces peristaltic movement. Inhibitory impulses are transmitted through fibres, which in the dog leave the cord by the anterior roots of the fifth to the eighth thoracic spinal nerves. These fibres thence pass into the sympathetic system, and ultimately reach the stomach by way of the splanchnic nerves.

*The duration of digestion* in health varies considerably according to the nature and quantity of the food taken, and also to individual idiosyncrasies. The normal limits lie between three and seven hours after a substantial meal, e.g. Riegel's test meal. A longer stay of food material in the stomach indicates motor insufficiency.

#### FUNCTIONS OF THE GASTRIC JUICE

Digestion commences in the stomach. In the mouth the food substances become mixed with the saliva, but then stay there is too short to allow of much digestive action taking place. The chief changes produced by the gastric juice are the following -

1 The soluble nutriment, which has escaped solution by the saliva, is dissolved.

2 The ptyalin of the saliva converts the starch into dextrins and maltose. This action continues as long as there is no free acid present in the stomach.

3 The acid of the gastric juice brings into solution any earthy phosphates or carbonates introduced with the food.

4 The gastric juice, in virtue of its pepsin and hydrochloric acid, dissolves insoluble and coagulated proteins and then allies, converting them into bodies resembling globulins. These proteins, which are already in solution, become chemically altered. The proteins undergo conversion into a body (acid albumin) precipitable on neutralisation, and not coagulable by heat. At a later stage bodies are formed which are not precipitable by neutralising the fluid, and as digestion proceeds the solubility of the bodies formed increases, and then precipitability by various chemical agents, e.g. saturation with neutral salts of the alkalis and magnesium, solutions of salts of the heavy metals, alcohol and strong mineral acids, diminishes. They also become more easily diffusible through animal membranes.

A chemical classification of the different products of gastric digestion has been founded upon these differences of solubility and precipitability. This classification is of considerable theoretical interest, but on account of the complexity and time-consuming nature of the methods employed for the complete separation of the different products, it has not, as yet, become of much importance in practical medicine. A very brief account of the most important facts must therefore suffice. The final product of gastric digestion is called peptone. The bodies representing the intermediate stages between it and acid albumin are called albumoses (or proteoses). The albumoses are all precipitable by saturation with ammonium sulphate, whereas peptone is not precipitable by this salt. Certain of the albumoses are also precipitable by saturation with sodium chloride or magnesium sulphate in neutral solution, and these have been named

the primary albumoses. The remaining albumose is not precipitable by saturation with sodium chloride in neutral solution, and it is named deuterio-albumose.

The pepsin and hydrochloric acid probably act as hydrolytic agents. This conclusion is supported (1) by the fact that the digestion of carbohydrates by the enzyme ptyalin is known to be hydrolytic, (2) by the fact that similar products to those of gastric digestion may be produced by boiling proteids with dilute mineral acids, which are known to act as hydrolytic agents, and (3) by the fact that peptone may be converted by certain dehydrating agents into bodies resembling native proteids.

With regard to the relative digestibility of different proteid and albumoid bodies, the article on diet should be consulted.

*Conditions affecting the rapidity of action of pepsin and hydrochloric acid.*—The most favourable degree of acidity is about 0.2 per cent hydrochloric acid. Other acids may replace the hydrochloric, but all of them are much less efficient.

The most favourable temperature for the action of pepsin lies between 35° C and 50° C. In neutral solution pepsin is destroyed by heating to 55° C, but the addition of peptones to the solution raises its resisting power, so that a temperature of 60° C becomes necessary for its destruction. In the dry condition it may be heated to a temperature of over 100° C without losing its activity. Up to a certain maximum an increase in the quantity of pepsin accelerates digestion.

Numerous organic compounds, which in dilute solution destroy, or at least inhibit, the activity of micro-organisms, exert little deleterious action on pepsin. Arsenious and salicylic acids, phenol, thymol, and chloroform in dilute solution only slightly retard peptic digestion. Neutral salts of the metals of the alkalis and alkaline earths lower the activity of pepsin. Alkalies and salts of the heavy metals rapidly destroy pepsin. It is of some importance to remember these facts in connection with the administration of drugs.

Accumulation of the products of digestion lessens greatly the rapidity of the action of pepsin. The duration of peptic digestion is thus greatly lessened by the simultaneous absorption of peptones by the stomach.

In virtue of the presence of the *rennin ferment* the gastric juice produces coagulation of the caseinogen of milk by converting it into casein. This action takes place in neutral solution prior to the appearance of free hydrochloric acid in the gastric contents. The caseinogen of milk is also precipitable as such by the acid of the gastric juices, and the precipitate is afterwards peptonised. The gastric juice splits up cane-sugar into dextrose and levulose. This action is said by some observers to be due to a ferment *invertin*.

Glucose and lactose are also said to be split up into lactic acid by means of an enzyme, even when bacterial action is excluded. This enzyme, however, has not been isolated, and its existence is doubtful. The quantities of lactic acid so formed are, in any case, so small that the occurrence of appreciable quantities is certainly to be regarded as pathological.

The hydrochloric acid of the gastric juice, in addition to its digestive action, acts as a *germicidal agent*. When the secretion of hydrochloric acid is deficient, the growth of micro-organisms occurs much more readily. Amongst these organisms may be mentioned the bacterium *lactis*, which by its action on carbohydrates produces lactic acid. Bacterial decomposition of proteids is also liable to occur.

*Examination of gastric contents.*—Methods used to obtain these.—The next point to consider is the examination of the gastric contents. It has already been mentioned that mechanical and electrical stimulation may be used to produce secretion of gastric juice. The most natural stimulus, however, is the introduction of digestible food material. The object of the examination of the gastric contents is to find out how the stomach fulfils its normal task of peptonising food material and propelling it into the intestines as the acid chyme. For this purpose a considerable variety of "test meals" has been employed.

The following are the more important ones—  
1 A test breakfast (Ewald and Boas), consisting of 70 gms white bread and 300-400 cc. of weak tea or water.

2 A lunch (G Sée), consisting of 100-150 gms bread, 60-80 gms of finely minced meat, and a large glass of water.

3 Riegel's test meal (given from 1-2 P.M.), consisting of a plate of soup (about 300 cc), a beefsteak of 150-200 gms, potatoes 50 gms, and white bread 50 gms.

Klempner, Bourget, Jaworski, and Gluzinski have also proposed test meals.

In considering the advantages and disadvantages of these meals, it will be found most convenient to take Ewald's breakfast and Riegel's dinner as types of the two extremes in quantity.

The chief advantages claimed for Ewald's method are the following—

1 The composition of the meal is definite and simple.

2 The siphoning off of the gastric contents can be undertaken after a short and definite interval (1 hour).

3 Practically every patient is able to take the meal on account of its small size.

Riegel's meal, on the other hand, is somewhat indefinite and complex in composition. It is difficult to fix upon the most favourable interval of time after the meal, for the obtaining of the gastric contents, as it varies in individual cases.

according to the degree of motor and peptic efficiency, from 2-6 hours. Usually the examination of the contents is undertaken  $3\frac{1}{2}$  hours after the meal. Lastly, the meal, on account of its large quantity, is not easily taken by all patients. Riegel's meal has, however, some advantages not shared by that of Ewald and Boas. In the first place, it lays a much greater demand upon the many-sided functional activity of the stomach, and therefore frequently gives one a better insight into what the stomach is really capable of performing. The results obtained by these methods do not always agree. The total acidity is usually found higher after Riegel's test meal than after that of Ewald and Boas. On the other hand, one must not omit to mention that in some cases, in which the production of hydrochloric acid is greatly diminished, *e.g.* in carcinoma ventriculi, free hydrochloric acid may be found after Ewald's test meal, whereas after that of Riegel a negative result of the examination is not infrequent. The explanation of this apparent contradiction lies in the fact that the large amount of pepsin in Riegel's test meal is able to enter into loose combination with all the hydrochloric acid secreted. Another important advantage is that it may be employed in the same way as the similar meal advised by Leube for testing the motor efficiency of the stomach. For general use, if only one method be employed, probably Ewald's is the more convenient.

The gastric contents may be obtained by means of a soft rubber tube or sound about 75 cm in length, and of variable diameter, 6-7 mm being the average, to whose upper end a small glass tube (about 4 inches in length) is connected by means of rubber tubing. The lower end of the sound may be closed or possess a terminal aperture, but in all cases should have one large or several small (Ewald and Rosenheim) lateral openings. The presence of the lateral openings lessens the risk of obstruction by food particles. The sound, having been rendered aseptic, is placed in warm water. In syphilitic, tuberculous, or cancerous cases, separate sounds ought to be used for each class of case. The upper glass end is then put into a vessel, which may be held by the patient. The lower end of the sound is next introduced into the patient's mouth, and pushed as far back as the root of the tongue (for this manipulation the introduction of the operator's finger is unnecessary). If the patient now swallow once or twice the tube will glide into the upper end of the oesophagus, its descent into the stomach being afterwards aided by slowly and rhythmically pushing it onwards until about 45 cm of the tube has been introduced.

For obtaining the gastric contents one of two methods—expression or aspiration—may be employed. The former method is the simpler and the one most frequently used. The patient

is requested to take a deep inspiration, and then by the contraction of his abdominal muscles to expel the gastric contents upwards through the sound into the vessel prepared for their reception. The index finger should then be placed on the opening of the glass tube and the sound rapidly withdrawn. The closure of the lumen of the tube by the finger prevents the escape of the fluid contained within the sound and rubber tube. By removing the finger the contents are then allowed to flow into the receiving vessel.

For the aspiration method, Boas' aspirator, or other similar instrument, may be employed. A description of the different forms in use is unnecessary, since the expression method has the advantage of greater simplicity, and is quite as efficient.

*II Clinical examination of the gastric contents*  
—A Macroscopic examination.—The chief points to note in the macroscopic examination are the quantity, consistence, odour, and colour of the contents, as well as the presence of any foreign constituents, such as bile, blood, abnormal quantities of mucus, etc.

Before forming an opinion with regard to the quantity, one must feel sure that the total contents have at least been approximately obtained. The quantity is, of course, largely dependent on the form of meal taken. No conclusion can be drawn from the quantity of the contents as to the peptic activity of the gastric juice. The digestive activity of the juice can be better ascertained from the other macroscopic appearances of the contents. From the quantity found, however, one can infer whether motor insufficiency of the stomach is present or not. In cases of increased motility the stomach may be found nearly empty after Riegel's test meal within a shorter interval than the normal one of seven hours. In other cases the quantity is abnormally large, or the stomach is found to still contain food residues after the lapse of seven hours. These are primarily cases of motor insufficiency. As will be seen later, motor and secretory insufficiency run by no means always parallel.

Lastly, in some cases a larger quantity is found than that which was introduced into the stomach. These cases were hard to explain according to the former view that water was absorbed from the stomach. More recently, however, von Mering has shown that not only is little or no water absorbed by the stomach, but, on the contrary, the absorption of certain substances, *e.g.* sugar, dextrin, peptones, and alcohol, is associated with a secretion of water into the stomach.

It is further important to note the presence or absence of undigested food residues, and also whether these food residues are chiefly carbohydrate or chiefly proteid in nature. A fine uniform liquefied mass suggests active peptonising power, accompanied, it may be, by hyper-

acidity. Undigested particles of food suggest the presence of subacidity. Abundant starchy residues associated with good proteid digestion justify the conclusion that hyperacidity is present. A certain opinion can of course only be formed with regard to the secretion after a detailed chemical examination. The necessity, however, of a careful preliminary macroscopic examination may be illustrated by the following examples.—In one case there may be found only a small quantity of hydrochloric acid, and yet the macroscopic examination may indicate a fairly complete putrefaction of the food. In another apparently similar case of subacidity, undigested food particles may be found in considerable quantity. If in these two cases an estimation of hydrochloric acid had alone been made, little or no difference in the conditions would have been detected. The consideration of the results obtained by macroscopic examination, combined with that of the results of the chemical examination, shows the important difference that in the former case the quantity of pepsin is much larger than in the latter.

The *odour* of the gastric contents is also of considerable significance. Normally they have no distinctive odour. In certain pathological conditions they have a somewhat rancid smell owing to the presence of free fatty acids. In cases of subacidity they sometimes have a putrefactive odour owing to the decomposition of proteins brought about by the growth of micro-organisms. In cases of intestinal obstruction or of an abnormal communication between the stomach and the intestines they may have a faecal odour.

Mucus in considerable quantities, blood, bile, and pus are abnormal constituents that may occasionally be found.

Mucus, if present in large amount, can easily be recognised by its physical characters, and by the fact that it yields a precipitate on the addition of a little acetic acid. It is usually found in the upper part of the gastric contents when they have been collected in a vessel. The quantity of mucus normally present is small.

*The Colour*.—Blood, when present, may produce either a reddish or brown tint in the gastric contents. In the former case it has been freshly shed, and microscopical examination frequently reveals intact red blood-corpuscles. In the latter the haemoglobin has become decomposed into methemoglobin and acid haematin (see "Hæmatemesis").

Bile and intestinal juice are not infrequently met with even under normal conditions, the bile giving a greenish tint to the gastric contents. The usual tests for its chief constituents may be employed in its recognition. The presence of intestinal juice is proved by testing for the pancreatic enzymes.

Pus is very rarely found. It may be recognised on microscopical examination, and by

means of the chemical tests used for its recognition in the urine.

In gaseous fermentation of the gastric contents the macroscopic appearances are very characteristic. The gastric contents, when collected in a vessel, show at least three distinct layers. The uppermost layer consists of foam formed by gas bubbles which rise to the surface, the middle layer is a more or less turbid fluid, and the lowest one consists of a sediment composed of fine carbohydrate residues. This condition is found under circumstances which allow stagnation of the gastric contents, as for example in motor insufficiency. The quantity of free hydrochloric acid is usually increased rather than diminished. In this respect gaseous fermentation is in striking contrast with other forms of fermentation, e.g. lactic acid fermentation, which are practically never found except in cases of subacidity. The mixture of gases present is occasionally inflammable. Chemical examination has shown the presence of variable quantities of the following gases.—Carbon dioxide, hydrogen, marsh gas, and acetylene, in addition to nitrogen and small quantities of oxygen. The last two gases are derived from atmospheric air which has been swallowed. Sulphuretted hydrogen is also occasionally found in the gastric contents, but it is not usually found in typical cases of gaseous fermentation.

For the examination of vomited material the same methods, macroscopic and chemical, are used as for the examination of the gastric contents obtained by the use of the sound. The vomited material may consist of mucus alone, mucus mixed with bile, or more or less digested food material with admixture of any of the pathological constituents already mentioned.

In certain cases the examination of the gastric contents after a test meal requires to be supplemented by a similar examination in a state of hunger, i.e. in the morning before breakfast. The presence of any quantity above 50 cc is to be regarded as pathological. Quantities above 50 cc are usually found in cases of motor insufficiency or obstruction, and secondly in cases of hypersecretion.

*B* The chief *morphological constituents* that may be noted on *microscopical examination* are the following.—Undigested food residues, e.g. muscle fibres, starch granules, plant cells, fat globules, etc., cell nuclei, squamous epithelium, columnar epithelium, mucous cells, blood and pus corpuscles, small portions of the gastric mucosa, particles of tumours, and various micro-organisms, e.g. yeast cells, sarcoma ventriculi, and numerous forms of bacteria.

*C. Chemical examination of the gastric contents.*—Prior to the chemical examination the gastric contents should be filtered. The filtrate alone is used for the examination.

1 The reaction of the filtrate should be

ascertained by the use of blue and red litmus papers.

2 If the reaction be acid, the presence or absence of free hydrochloric acid should be ascertained. There are a number of organic colouring matters, which change in colour in the presence of free hydrochloric acid even when the acid is in very dilute solution, while they show no change of colour in presence of such strengths of organic acid solutions as are ever found in the gastric juice. The most commonly employed colouring matters are tropæolin OO (saturated alcoholic solution), which is changed from yellow to red by free hydrochloric acid, methyl violet (1 per cent aqueous solution), which becomes blue, and congo red, which is converted into a dark blue. The most important objection to the use of all these is that albumoses and peptone when present prevent or lessen the colour change by entering into loose combination with more or less of the hydrochloric acid. This objection does not hold good for Gunzburg's test. Gunzburg's reagent has the following composition—Phloroglucin 2 gms., vanillin 1 gm., absolute alcohol 30 cc. A few drops of this yellowish fluid are mixed in a porcelain basin with a very small quantity of the filtrate, and the mixture evaporated to dryness over the open flame. If free hydrochloric acid be present, a carmine red residue is left, but if free organic acids only be present, a slight yellowish residue is left.

The simplest test for lactic acid is Uffelmann's reagent (2 per cent aqueous phenol solution to which are added a few drops of tincture of the perchloride of iron until the fluid becomes of a deep amethyst-blue colour). This reagent should always be freshly prepared before use. It is not advisable to test for lactic acid by its means in the filtrate itself, as other substances, which share with lactic acid the power of converting the blue into a canary yellow solution, may be present in the filtrate. Amongst these may be mentioned glucose, phosphates, alcohol, oxalic, citric, and tartaric acids. About 10 cc of the filtrate are concentrated to a thin syrup by evaporation on the water bath, thoroughly extracted with ether, using about 50 cc. in all. The ethereal layer is then separated, and evaporated to dryness on the water bath. The residue is dissolved in water, and tested by means of Uffelmann's reagent. For a quantitative estimation of lactic acid the extraction with ether must be more thorough. At least six extractions, using 100 cc of ether each time, should be employed. The residue after evaporation of the ether should be dissolved in a known quantity of water and titrated with decinormal caustic soda solution. Each cc of the decinormal alkali is equivalent to 0.090 gms. of lactic acid.

Butyric and acetic acids are also sometimes found in very small quantity.

The filtrate of the gastric contents may also be tested for the different forms of albumoses and for peptone, but as yet the examination has not obtained much practical importance.

The presence of pepsin may be recognised by means of an artificial digestion. Ten cc of the filtrate are placed in a test-tube, hydrochloric acid being added, if necessary, until congo red paper is just turned blue, and a circular disc of hard-boiled white of egg 1.5 mm thick and 10 mm in diameter is placed in the fluid. The disc may be cut from a hard-boiled egg by means of a Valentine's double-bladed knife and a cork-liner. The test-tube is then placed on a water bath at 40° C. If the quantity of pepsin be normal, the albumin disc should be completely dissolved in a half or at most a whole hour.

To test for the presence of rennin, 10 cc of the gastric filtrate are accurately neutralised by the addition of decinormal caustic soda solution. A few drops of the neutral fluid are then added to 10 cc of milk in a test-tube, and the mixture is placed in a water bath at 40° C. If rennin be present in sufficient quantity, coagulation of the milk should be complete in ten minutes, or at most half an hour.

*Quantitative estimations of the chief constituents of the gastric filtrate.*—

The total acidity may be estimated by the following method.—To 10 cc of the gastric filtrate, diluted with a little water, a few drops of alcoholic solution of phenolphthalein are added. Decinormal caustic soda solution is then gradually added to the mixture placed in a beaker until a permanent pink colour just forms. It is customary to express the degree of acidity as found by a figure stating the number of cc of decinormal caustic soda necessary to neutralise the total acid in 100 cc of the filtered gastric contents. If, for example, 5.7 cc of decinormal caustic soda were required to neutralise the acid in 10 cc of the gastric filtrate, then 57 cc would be required to neutralise the acid in 100 cc, and the acidity would be 57 per cent.

Other indicators, *e.g.* litmus, may be used in the place of phenolphthalein. The indicator chosen should be employed for all the estimations of one series. This is a necessary precaution, as the results for the acidity obtained by the use of different indicators do not always coincide when proteids are present in the acid fluid. If, for example, a drop of phenolphthalein solution be added to a proteid one, which reacts neutral towards litmus, the addition of a small quantity of decinormal caustic soda will be found necessary before a permanent pink colour of the solution is secured. The total acidity varies within wide limits even under physiological conditions. 50-75 per cent may be given as the average.

The total acidity of the gastric filtrate is

made up of the following factors—(1) free hydrochloric acid, (2) combined hydrochloric acid, (3) acid salts, and (4) organic acids. The quantity of organic acids present is usually so small that it may be neglected. A method for the estimation of the most important one of these, namely, lactic acid, has already been given. For methods of estimating the other organic acids see "Literature."

Probably the simplest and yet accurate method of estimating the free hydrochloric acid is Fleiner's modification of that of Mintz. To 10 cc of the gastric filtrate placed in a beaker are added about twenty-five drops of Gunzburg's reagent. A drop of the mixture is cautiously evaporated to dryness over the open flame. A carmine-red residue indicates the presence of free hydrochloric acid. For the quantitative estimation of the free mineral acid decinormal caustic soda is gradually added from a burette until a drop or two of the mixture yields no red residue on heating. After making a preliminary estimation a second one should always be performed, using the result of the first as a guide. The number of cc of decinormal caustic soda used indicates the quantity of free hydrochloric acid expressed as so many cc of a decinormal hydrochloric acid solution. The percentage of hydrochloric acid is obtained by multiplying this result by 0.0365. Although this is always given as a method for the estimation of the free HCl alone, it is doubtful whether, after all, some at least of the combined HCl is not also estimated.

The acidity due to acid salts may be estimated by Leo's method. 10 cc of the gastric filtrate are mixed with 5 cc of a saturated solution of calcium chloride, and the fluid titrated with decinormal caustic soda in precisely the same manner as already described under estimation of the total acidity.

To 15 cc of the gastric filtrate is then added 1 gm of finely powdered pure calcium carbonate. The mixture is thoroughly shaken, and then filtered through a previously dry filter into a well-dried beaker. Calcium chloride, acid phosphate, the excess of calcium carbonate, and carbon dioxide are present in the filtrate. The carbon dioxide is removed by passing a stream of air through the fluid by means of a hand bellows connected with a fine glass tube dipping into the fluid, or by means of an aspirator. 10 cc of the filtrate, now freed from carbon dioxide, are accurately measured out, mixed with 5 cc of a saturated solution of calcium chloride, and titrated with decinormal caustic soda as before. The latter result gives the acidity alone due to acid salts. The former result gives the total acidity.

A few words are necessary to explain the principles upon which Leo's method is based. Solutions of free acids on being shaken with calcium carbonate in the cold are completely neutralised, while fluids containing acid (re-

dihydrogen) phosphates after similar treatment retain their acid reaction. In a mixture of free acids and acid phosphates, one can therefore exclude the acidity due to free acids by the addition of calcium carbonate, and then estimate in the filtrate the acidity due to acid phosphates. The soluble calcium salts which are formed by the interaction of calcium carbonate with the free acids present (chiefly hydrochloric) react with sodium dihydrogen phosphate to form sodium chloride and calcium dihydrogen phosphate. One then estimates the acidity of the latter salt, which requires twice as much caustic soda to convert it into the neutral calcium phosphate as is necessary for the neutralisation of the corresponding acid phosphates of sodium and potassium. The following equations will help to render the matter clear— $2\text{NaH}_2\text{PO}_4 + 2\text{NaOH} = 2\text{Na}_2\text{HPO}_4 + \text{H}_2\text{O}$ , whereas  $2\text{NaH}_2\text{PO}_4 + 4\text{NaOH} + 3\text{CaCl}_2 = \text{Ca}_3(\text{PO}_4)_2 + 6\text{NaCl} + 4\text{H}_2\text{O}$ .

To overcome this difficulty, one of two expedients may be employed. The total acidity may be estimated in the usual way without the addition of calcium chloride. The second titration may be carried out exactly as before. The number of cc of caustic soda used in the second titration would, however, require to be divided by two. This division by two becomes unnecessary if both estimations are performed under the same conditions, namely, the addition of excess of calcium chloride. Since the second method is the simpler, it is almost invariably employed.

The total quantity of hydrochloric acid—free and combined—is easily ascertained from the results already given. It is equivalent to the total acidity minus that due to acid phosphates and lactic acid if present.

For the estimation of the total "physiologically active" hydrochloric acid (free and combined) numerous other methods have been employed, of these Martins and Luttke's appears to be the most accurate. As the method, however, is somewhat too complex for clinical use, a brief sketch of the principles upon which it is founded must here suffice. The total chlorides in 10 cc of the gastric filtrate are first estimated as in the urine by Volhard's method. The quantity of chlorine in the ash of another 10 cc is then estimated and reckoned in terms of hydrochloric acid. By subtracting the latter result from the former, one obtains the chlorine present as free and combined hydrochloric acid.

The loosely combined hydrochloric acid is equivalent to the total acidity minus that due to acid phosphates, free hydrochloric acid, and lactic acid if present. The combined hydrochloric acid is in loose union with amino-acids, albumoses, and peptones. The total hydrochloric acid is sometimes termed the "physiologically active" acid, because the loosely combined is as important in peptic digestion as the free

THE MOTOR ACTIVITY OF THE STOMACH may be tested by several methods. 1 Probably the best of these for practical purposes is that of *Leube*. Seven hours after the taking of a test meal similar to that of Riegel, an examination is made with the gastric sound. Under normal conditions the stomach should then be found empty, but in cases of motor insufficiency the amount of fluid varies according to the severity of the case.

2 The *salol method* of *Erwald and Sievers* for testing the motor efficiency of the stomach is founded upon the fact that salol—a compound of salicylic acid with phenol—is not decomposed in acid solution, but readily undergoes decomposition in alkaline solution into phenol and a salicylate of the alkali employed. This decomposition cannot take place in the stomach, but occurs whenever the salol passes into the small intestine. The salicylate thus formed is absorbed, and excreted in the urine as salicylic acid, which gives a violet colour with ferric chloride. If 1 gm. of salol be given to a healthy individual during digestion, the reaction with ferric chloride should appear in the urine after half or at most three-quarters of an hour. Delay in the appearance of the reaction indicates a retention of the contents of the stomach. The salol test has, however, turned out to be far from reliable. Stern has shown that salol is absorbed from the stomach itself, and that the products of its decomposition may afterwards be detected in the urine.

3 A third method—that proposed by *Klemperer*—consists in introducing into the stomach, previously freed from food residues, 100 cc. of olive oil. After two hours the gastric contents are obtained by means of the sound, the complete removal of the oil being afterwards ensured by repeatedly washing out the stomach with water. The oil, after being separated from the water in a separating funnel, is measured. The diminution in quantity expresses the amount of oil which has been expelled into the intestines. *Klemperer* found that under normal conditions 70-80 cc. of oil were expelled into the intestines. This method, although of considerable scientific value, is obviously one ill suited for practical use.

4 Lastly, a short reference to *Einhorn's* electrical method for the examination of the motor activity of the stomach must suffice. For this purpose he uses an ingeniously contrived instrument called the gastograph. Although indicating all movements of the gastric contents, it is chiefly of use as a recorder of those gastric movements which complete the mechanical subdivision of the food and ensure its thorough admixture with the gastric juice. The method has not so far proved of much practical utility.

*Pathological variations of gastric digestion* may be divided into secretory, motor, and sensory.

The variations in secretory activity may be either in the direction of increased or of diminished activity, and may affect one or all of the constituents of the gastric juice. Whereas under normal conditions the gastric mucosa only secretes in consequence of some stimulus, in hypersecretion it continues to act during the intervals between meals, and, in consequence, the stomach is found to contain fluid even during hunger. This hypersecretion or gastrosuccorrhoea may be intermittent or chronic, and is usually associated with more or less sensory disturbance.

Diminished secretion of gastric juice is found, especially in atrophic conditions of the gastric mucosa, e.g. in atrophy of the ventricle.

Anomalies of secretion affecting individual constituents of the gastric juice are usually limited to variations in the quantity of hydrochloric acid produced, the quantity of pepsin remaining fairly constant, and may manifest themselves as either a subacidity or a hyperacidity of the gastric juice. The total acidity in such cases may reach values of from 100 to 150 per cent, while the acidity due to free hydrochloric acid may amount to 50 to 60 per cent. The alterations in acidity in the different pathological conditions affecting the stomach (carcinoma, gastric catarrh) are referred to under their special headings.

Diminished motor activity of the stomach may affect the muscular walls of the organ as a whole, e.g. in atony of the stomach, or be limited to certain parts, as in pyloric or cardiac insufficiency.

Increased motility may be due to increased resistance to the expulsion of the contents as in pyloric stricture, or it may be established to compensate for secretory insufficiency, or it may be due to irritation, as in hyperchlorhydria, or lastly it may be nervous in origin. It may be general, as in peristaltic unrest of the stomach and in vomiting, or it may take the form of spasmodic contractions localised to the cardiac or pyloric orifices.

While digestion is normally unaccompanied by sensation, in pathological conditions the varieties of sensory disturbance that may occur are so numerous that reference must be made to the special articles on diseases of the stomach.

#### BILE

The secretion of bile takes place continuously, in this respect differing from the true digestive secretions. At the present time bile is regarded mainly as a fluid which contains certain effete products destined for excretion, some of these, however, being absorbed by the intestine after their discharge from the bile duct. Certain substances which are insoluble in such a watery solution as urine can be dissolved and so excreted by the bile. The most important biliary constituents are formed in the liver, not

being found in the blood either normally or after removal of the liver, but if the outflow of bile be hindered, the biliary constituents appear in the blood and are partly deposited in different tissues, partly excreted by the kidneys, giving the urine a brownish-green colour.

**PHYSIOLOGICAL ACTION.**—Until comparatively recently it was assigned a very important rôle in digestion, but now it is only regarded as of importance in the absorption of fats. It does not exercise any direct deterrent action on putrefying processes so far as can be made out. The digestion of proteids and carbohydrates is practically unaffected by its withdrawal, but only about half the normal amount of fat is absorbed, the rest being excreted in the faeces. This withdrawal of fat in the faeces allows the action of putrefaction organisms on the proteids, because the latter become coated with fat, and so the action of the pancreatic juice is prevented, and decomposition ensues. So long, however, as excessive quantities of fat in the food are avoided, neither the general condition, *e.g.* nitrogen equilibrium, nor digestion suffers. It is exceedingly difficult to study the action of drugs on the bile secretion, because the flow is such a variable one even under ordinary conditions, and as a result of this the conclusions which different investigators have come to are very contradictory. It seems probable that no substance, with the exception of certain of the bile constituents themselves, acts as a true choleragogue. The pressure of the bile in the ducts can rise to a much greater height than that of the blood in the portal system. The temperature of the blood in the hepatic vein is higher than that in the portal, showing that the cells have been undergoing active metabolic changes, but of course this applies not only to the biliary, but also to the glycogenic function of the liver.

**MODE OF ORIGIN OF CONSTITUENTS.**—The only bile constituent of which we know more or less definitely the mode of origin is the bile pigment. This pigment occurs only in animals which contain haemoglobin in their blood. It is certain that the blood pigment is either set free in the liver from the breaking down of red blood-corpuscles, or it is set free elsewhere and carried to the liver, where it is split up into an non-holding radicle, haematin, and albumin. The former loses its iron and takes up water to form bilirubin, the iron being retained in the liver, perhaps helping to form haemoglobin later.

A very small quantity of iron is excreted in organic form in the bile, not nearly sufficient to account for the amount present in the haemoglobin before destruction. After injection of haemoglobin into the blood, there is an increased production of bilirubin, but this does not result in an absorption and deposition of pigment until the amount becomes so great that the bile capillaries or smaller bile ducts become blocked. Any agents which can break down

the red blood-corpuscles setting free haemoglobin can produce bilirubinuria if the amount of disintegration be great enough to produce obstruction in the bile capillaries. On obstruction to the bile outflow, the resultant absorption takes place in the larger bile ducts, through the lymphatics into the thoracic duct. Bilirubin can be formed from haemoglobin outside the liver, as, *e.g.*, in old blood extravasations, hæmorrhagic infarcts, etc. The pigment which crystallises out under such conditions was first of all described by Viechow as hæmatoidin, but it is now known to be identical with bilirubin. There are many other facts which bring out strongly the intimate connection between the blood and bile pigments. Only one of these need be referred to, namely, the formation and excretion in the urine of the important iron-free blood pigment, hæmatoporphyrin, in certain conditions, *e.g.* after sulphonal poisoning (see "Hæmatoporphyrin"). This pigment is isomeric with bilirubin. The chief pigment in human bile is not, however, bilirubin, but biliverdin, which gives the green colour of the fresh biliary secretion. It is rapidly reduced in the intestine, from the action of bacteria, into urobilinogen and derivatives of that pigment. The source of the other constituents of the bile has not been so well made out. *Cholesterol*, a constituent probably of every living animal cell, is, like the bile pigment, an effete product. It is derived from the metabolism of the cell protoplasm, and is distinguished by the resistance which it offers to further decomposition, and also by its tendency to form gall stones. With regard to the salts of the bile acids, glycocholic and taurocholic, they are the only bodies which can undoubtedly act as chologogues, undergoing absorption by the intestinal mucous membrane after their excretion into the duodenum. There can be no doubt, however, that this absorption takes place, not in the duodenum, but in the jejunum and ileum. As these acids undergo partial absorption, alterations in the amount of proteid in the food do not affect to any extent their excretion in the bile. Thus, after increasing the amount of proteid in the food, the salts of the bile acids are barely increased at all in the bile. Their absorption may take place directly into the blood-vessels of the intestinal mucosa, although probably the lymphatics form the main channel. These salts probably play an important part in aiding the absorption of other substances by the intestine—for example, soaps of the alkaline earths and fats. The other constituents of the bile are not of so much importance. The body which lends the peculiar stringy consistence to the bile, namely, *mucin*, is secreted by the gall bladder and larger bile ducts. In man it is a true mucin, but in the ox it is supposed to be a nucleo-albumin. There are also present in the bile small quantities of fat, *lecithin*, and soaps.



The amount secreted can only be estimated in cases of biliary fistula, and such estimations are not reliable, because a certain amount of the bile is normally absorbed by the intestine, and acts upon the biliary function of the liver. If it be then withdrawn by means of a fistula, this natural chologogue is removed, and so less is excreted than what would normally be the case. The quantity also varies with the amount of water taken and the nature of the diet, animal food increasing it to the most marked extent. The maximum of flow occurs some hours after food has been taken, the larger the meal, the longer the interval before the maximum appears. Exceedingly little is known as to the action of the nervous system on bile secretion. There seem to be certainly no true secretory nerves, but the secretion may be affected by vaso-motor influences. If the blood of the portal vein be allowed to pass directly into the inferior vena without passing through the liver, the secretion of bile still goes on, and the same holds good after obstruction of the hepatic artery. After splanchnic stimulation there is a diminished flow, with contraction of portal vessels, while after section the opposite occurs. The expulsive action of the gall bladder and larger bile ducts is under the influence of the nervous system, peristaltic waves of contraction, similar to the intestinal ones, occurring along their smooth muscular fibres. These may also be stimulated reflexly from the gastric mucous membrane. The removal of bile from the liver through the ducts is caused by the back pressure of the newly formed secretion, and also from the pressure on the liver during inspiration.

**PATHOLOGICAL ALTERATIONS**—It may undergo *pathological alterations*, of which the chief are due to obstruction to the bile outflow when the pigments and salts of the bile acids are absorbed, and the fluid left in the dilated channels is often found to contain only traces of the true constituents with an excess of mucus (see "Jaundice").

Under certain conditions, the nature of which we do not yet know, *gall stones* may form in the ducts. These vary in size, form, and chemical composition. There are two important forms met with in man, cholesterol and pigment chalk stones, the former being by far the more frequent. They are white or faintly yellow in colour, and are specially characterised by their low specific gravity, which is less than that of water. Absolutely pure cholesterol stones are rare, there being usually a bilirubin chalk nucleus around which the cholesterol has crystallised out. The pigment stones are much more easily broken down by pressure than the others, and are usually much smaller and heavier. They very often contain traces of copper and a larger or smaller quantity of other bile pigments in addition to bilirubin, e.g. biliverdin. When numbers of gall stones have collected in the

ducts, they present a faceted appearance, owing to the pressure to which they have been subjected. Very often small stones or gravel, composed of calcium carbonate tinted with bilirubin, are passed. The occurrence of traces of heavy metals, e.g. Ca, Zn, As, emphasises the importance of the bile as a medium for excretion. With regard to the causes leading to the formation of gall stones, it has been surmised that in the case of cholesterol ones, two factors may have played a part, viz. insufficiency of bile salts to keep the cholesterol in solution, or excessive formation of the latter. The most probable cause leading to the formation of both kinds of calculi is obstruction to the bile outflow owing to catarrhal conditions of the ducts, when the excessive secretion of mucus or the shedding of epithelium may at least aid in their production.

**PANCREATIC SECRETION.—NATURE AND METHOD OF SECRETION**—The most important changes which the food-stuffs undergo are the result of the action of the *pancreatic juice*. The acid chyme is gradually neutralised by the clear alkaline fluid which is secreted by the cells lining the pancreatic acini, and which is poured into the duodenum through Wirsung's duct. If the fluid be obtained from a fistula, it is found at the outset to have a specific gravity of about 1.03, but, after some time has elapsed, it becomes poorer in proteins and of lower specific gravity. In carnivora and omnivora the secretion is not a continuous one. During activity the cells of the pancreatic acini undergo distinct alterations. Thus at the beginning of digestion the granules of zymogen gradually disappear from the inner zone until the whole cell at last appears clear. Then the granules are again formed in the inner part, the outer zone acting as the storehouse for the material out of which the zymogen is formed. These zymogen granules are only transformed into the active ferment after the gland has been exposed to the air for some time, or after it has been subjected to the action of weak alkalis or acids. The amount of the juice secreted in twenty-four hours is impossible to estimate accurately, as in cases of fistula it undergoes marked alterations, rapidly also becoming poorer in solids. As in other similar secretory processes, the secretion is accompanied with vascular dilatation.

**NERVOUS MECHANISM**—If the vagus be stimulated, there is an increase in the amount secreted, the fluid also being thick in consistence, while, if this nerve be cut, and its central end stimulated, the flow is greatly diminished, or may be stopped altogether. A rather important cause of stoppage of secretion is vomiting, here also the influence of the vagus making itself felt. It is an interesting fact that the acid chyme, on coming into contact with the duodenal mucous membrane, causes an increased pancreatic secre-

tion, and this brings about neutralisation, while alkalies have the opposite effect. This reflex secretion persists even after the vagi and sympathetic have been cut, its occurrence being then probably due to the presence of a local centre perhaps in the neighbourhood of the pylorus. The maximum flow occurs about three hours after food has been taken, then sinks for about the following four hours, when it may again rise. The greater the flow, the poorer is the juice in solids.

**ACTION OF THE ENZYMES.**—*The ferments in the pancreatic juice* are four in number, the most important one being *trypsin*, which acts upon proteins, transforming them into more soluble bodies.

Unlike pepsin, it acts best in a weakly alkaline solution, although it is also active in neutral or faintly acid ones. Still even with weak organic acids, although at the outset the action is almost as marked as in slightly alkaline media, the ferment is slowly destroyed, disappearing, however, much more rapidly with weak inorganic acid solutions. Thus it is useless giving such preparations as pancreatin by mouth, because trypsin is rapidly destroyed by the acid gastric juice. The first action which it exercises on proteins is simply a solvent one, and it is doubtful whether any alkali-albumin is found or not. The digestion passes so rapidly through the stage of primary albumoses that it is often difficult to detect their presence, but deuto-albumose (a secondary one) is formed in large quantity, and then peptone makes its appearance. But the most characteristic feature, from the chemical standpoint at least, in tryptic digestion is the further decomposition of the peptone. Until recently it was believed that all the peptone did not undergo decomposition, but that a part was resistant, this being termed antipeptone, while the rest, which was split up into amido-acids (such as leucine, tyrosine, and aspartic acid), ammonia, etc., was termed henipeptone. Siegfried regarded antipeptone as identical with an acid which he found in paired combination in muscle and milk, namely, carnine acid, but it seems unlikely that this is the case, as Kossel, Kutscher, and others have shown that Kuhne's antipeptone is really a mixture containing certain bases, called the hexone bases (lysine, histidine, and arginine). These bodies are only formed after very prolonged digestion. The point of importance is that, in all probability, if only digestion be continued long enough, all the peptone can be broken down. Now, although this is true for artificial tryptic digestion, the action is a much more limited one in the intestine, very little leucine and tyrosine ever being formed under normal conditions. In fact, it is probable that the pancreatic juice is mainly required to render the proteins soluble and ready for absorption in cases where such large quantities have been taken that, if this action

did not take place, a great loss of protein would result, this material undergoing decomposition in the lower parts of the intestinal canal. As we shall describe more fully later, one is compelled to admit that even the transformation into albumoses and peptones is not absolutely essential because the forerunners of these (soluble albumins and albuminates) are themselves capable of absorption by the intestinal mucosa.

The products of a pancreatic digestion easily undergo putrefaction, and hence in artificial digestive fluids such an agent as thymol or chloroform requires to be added. In addition to the substances mentioned above, a chromogen termed tryptophan, the nature of which has not yet been fully made out, is formed about the time that the amido-acids appear. A small amount of free ammonia also appears during digestion. Such is very shortly the nature of the hydrolytic decomposition of a simple protein under tryptic digestion. Complex proteins are also more energetically attacked by trypsin than pepsin. Thus nucleo-albumins have not only their albuminous constituent easily broken down, but the resistant nucleic radicle is also slowly dissolved.

There are in addition three other enzymes which occur in the pancreatic juice, an amylolytic, a fat-splitting, and a milk-curdling one. The *amylolytic* ferment, so far as we at present know, acts on the polysaccharides in the same way as ptyalin. Thus starch and glycogen undergo hydration and are split up into the dextrins, which were described under salivary digestion, and maltose. The amount of the final product, maltose, depends on the relative quantities of enzyme and polysaccharide. The monosaccharides undergo no alteration, but are absorbed without alteration, while cellulose is not affected by the juice.

The action of the *pancreatic juice on fats* is a more complex one. The acid chyme is neutralised by the bile, pancreatic juice, and the secretions from the intestinal glands, and then, if fatty acids be present, emulsification of fats can at once take place, but the fat-splitting ferment assists in this action, setting free fatty acids from the neutral fats. This fat-splitting action does not require to be a very marked one, because a very small quantity of free fatty acid is able, in the form of a soap, to emulsify a large quantity of fat. Later on, the mode of absorption of the emulsion will be referred to. Lecithin is also split up by this ferment. Bile seems to assist this ferment in its action. Like all other enzymes, it is easily destroyed by boiling, and the same occurs if the gland be exposed to the air for a short time, as in the artificial preparation of pancreatic extracts.

*The milk-curdling ferment* of the juice seems to be identical with rennin, only its mode of action has not been so carefully worked out as that of the ferment derived from the stomach.

**PATHOLOGICAL ALTERATIONS** in the juice have not been much investigated. In certain inflammatory conditions affecting the pancreas the juice secreted is thin and inactive, and in other pathological conditions there may be a stoppage of the flow, due to the ducts being blocked up. The pancreas, however, plays an important part in the metabolism of glucose, which may be referred to here. If the gland be removed entirely from dogs, diabetes of a severe type at once sets in, and death ensues in a short time, but if a small portion be left, so long as this does not undergo atrophy, only traces of glucose appear in the urine. As soon as the remaining piece atrophies, marked glycosuria sets in, and within a few weeks or months the animal dies. In such cases, glucose is excreted even when no carbohydrate is given in the food, and if any be given by the mouth, it is wholly excreted in the urine. The blood in such cases shows a high percentage of glucose—0.3-0.5 per cent. It is important to note that mere withdrawal of the juice from the intestine does not produce diabetes, nor does ligation of the pancreatic vein. At present we do not know in what way the pancreas exercises an influence on the metabolism of glucose. In a few cases of diabetes the pancreas has, on post-mortem examination, been found to be atrophied, or otherwise pathologically altered (see "Diabetes").

**INTESTINAL SECRETION.**—The succus entericus is secreted by Lieberkuhn's glands of the small and large intestines. It is obtained usually from fistulae (Thiry-Vella), when a piece of small intestine, about 50 cm. long, has been resected with the mesentery attached and the upper and lower openings sewed into the abdominal wall.

It is a clear yellow fluid of markedly alkaline reaction, containing usually a fairly large quantity of solids, although the amount of these varies very much. With regard to the *enzymes* present there is a great deal of contradictory evidence. It seems most likely that it contains no ferment which acts on proteins or fat, but it certainly contains an inverting ferment, and very probably more than one. In some animals the secretion appears to be a continuous one, in others it only occurs after stimulation of the mucous membrane. Experimentally one may use either mechanical, chemical, or electrical stimuli. Usually the amount secreted begins to increase about one hour after food, and gradually rises to about the third hour, when it falls again. Very little is known as to *nervous influences* exerted on this secretion. If all the nerves, passing to such an intestinal loop as has been described above, be cut, fluid accumulates in the sac. Stimulation or section of the vagi seems to have no effect. With regard to the action of the juice on the food, there seems always to be a slight diastatic action, starch being converted into maltose, but more important is the inverting action trans-

forming maltose into glucose and cane-sugar into glucose and levulose. Lactose does not seem to be inverted by the juice, although it may be in its passage through the intestinal mucosa. The inverting action takes place in the presence of antiseptics, but is destroyed by boiling.

*In the lower part of the small intestine*, where putrefactive organisms possess a suitable slightly alkaline medium, the proteins undergo changes which are at the outset similar to those produced by the gastric and pancreatic secretions. That is to say, insoluble albumins are transformed slowly into soluble forms (albumoses and peptones), and in all probability this action is due to enzymes secreted by the bacteria. These organisms do not attack unaltered albumin in the upper part of the intestine, because there they have a supply of albumoses and peptones ready for assimilation. Recently an elaborate series of experiments has been carried out in order to discover whether micro-organisms are necessary in intestinal digestion, and it has been shown that guinea-pigs which had been born in a sterile medium could live there even when their food was absolutely free from organisms. This of course does not touch the question as to whether bacteria may not be of use in aiding the action of the intestinal enzymes. And again there are substances present in the usual diet which are unaffected by the other enzymes, but are dissolved by bacterial action, *e.g.* celluloses. These micro-organisms can also break down proteins along different lines from the proteolytic enzymes. In the first place the aromatic radicle may undergo a variety of decompositions. Thus tyrosin is not only formed itself, but from it other benzol derivatives are obtained by reduction or oxidation, ending with the production of phenol, and in addition there are produced aromatic substances of a different order which are not found in ordinary tryptic digestion, namely, indol, skatol, and derivatives of these. In the second place, bodies of the fatty series are formed often in fairly large quantity, *e.g.* leucine and volatile fatty acids such as butyric, valeric, and caproic acids. There is no free oxygen in the intestine, owing to the continual formation of hydrogen and sulphuretted hydrogen from bacterial action, so that any parasites which are present must get it indirectly from the capillary blood of the mucosa by keeping as close as possible to the intestinal wall. The amount of oxygen which they require is very small.

*Ptomines.*—There are also bodies of a basic nature derived from the decomposition of proteins which normally are not formed in the intestine during life. These bodies, termed ptomines, do not appear until about three days after death. They are formed also when the intestinal bacteria are grown in different culture media. As a rule these bodies, which belong usually to the fatty series, are not very poisonous if they be pro-

duced rapidly after death, but those which make their appearance later are often extremely poisonous even in minute doses. The best examples of the non-poisonous class are *cadaverin* and *putrescin*, while *neurin*, derived from the decomposition of lecithin, is the best example of the poisonous series. This body is formed, unlike the ordinary poisonous ptomaines, comparatively rapidly after death, usually about the third day. One must bear in mind the possibility of the formation of such bodies in tinned meats and decomposing flesh of any kind, and also that similar bodies may be formed even during life in certain animals, e.g. mussels (see "Diet"). It is a comparatively rare occurrence to meet with the formation of those bodies under pathological conditions in the organism, but they do occur in the feces in cholera, especially *putrescin* and *cadaverin*. The primary poisonous products of bacteria are usually of the nature of globulins or albumoses.

Not only are proteids acted upon by micro-organisms in the lower parts of the intestinal canal, but fats are split up into glycerine and fatty acids, and carbohydrates undergo manifold fermentations or are simply hydrated. So long as there are carbohydrates present in the small intestine which have not undergone fermentation, the proteids are protected from the action of micro-organisms. Thus a milk diet, from the presence of lactose, prevents decomposition of proteids owing to the formation of lactic acid. The influence which unabsorbed fats exercise on the putrefaction of proteids has already been referred to. In the comparatively short small intestine of carnivora, and even in that of omnivora, very little putrefaction is set up compared to that occurring in the large intestine. Putrefaction in both small and large intestines is limited, however, to a comparatively small amount of the food-stuffs present, because, in the first place, micro-organisms, by rendering these substances more soluble, transform them into material which is more easily absorbed, and when they do break down proteids further, they form small quantities of substances, such as phenol, which check putrefaction, while in the last place, owing to the great absorption of water in the large intestine, the conditions under which the bacteria grow are rendered less suitable. Before taking up the subject of intestinal movements, etc., it is necessary to refer shortly to the modes of absorption of the digested material, and also to the nature of its transmission through the organism.

**Absorption.**—The part which the gastric mucous membrane plays in absorption is still doubtful. Although very little water is absorbed, a fairly large quantity of soluble salts, albumose, sugar, alcohol, etc., is taken up, but the main channel of absorption is through the intestinal mucosa. The amount of a food-stuff absorbed is independent of its osmotic equivalent.

**Absorption and Metabolism of Proteids.**—Although the absorption of proteids is rendered an easier and more rapid process by their peptonisation, there can be no doubt that soluble albuminous substances, such as syntonin and other albuminates, can be absorbed without undergoing further alteration. Modern treatment by rectal feeding is based on this fact, for in the large intestine the transformation of proteids into albumoses and peptones takes place only to a slight extent, not nearly sufficient to account for the fact that patients can by this means be kept in nitrogen equilibrium. This is true not only for the absorption, but also for the assimilation of soluble albuminous substances. Thus one can inject the blood-serum of another animal into the veins of a dog without albumin afterwards appearing in the urine. The formation of large quantities of albumose or peptone seems in fact to be deleterious, as the intestinal mucosa is apt to suffer from excessive stimulation. When albumoses or peptones are absorbed, they are not carried as such to the tissues, but undergo polymerisation into albumin in the intestinal mucosa.

Neither albumoses nor peptones are present in the blood at any time during digestion, nor are they stored up in the intestinal wall, and yet if they be allowed to lie in a separated intestinal loop they rapidly disappear. They must then undergo some alteration in the intestinal wall, and if the blood in the mesenteric or portal vein be examined after a meal rich in proteids, it is found to be much richer in albumin than before digestion, while the lymph in the thoracic duct shows no alteration in its percentage of protid. Ligation of the thoracic duct does not prevent the absorption of protid, nor the subsequent rise in the excretion of nitrogen in the urine. These facts go to prove that the absorbed albumoses are transformed into albumin, and that the channels into which the latter pass are the blood and not the lymph vessels. The means by which this transformation has been brought about are unknown. The leucocytes certainly do not seem to take up the albumose and peptone and transform them into albumin, because no evidence has been brought forward that they are capable of effecting such a transformation. The great mass of absorbed protid seems to act as a source of energy without being taken up by the tissues and organised, although a small amount must supply the place of the cell proteids, which are always undergoing degeneration. The amount of nitrogen excreted during the day in the healthy adult is equal to the amount absorbed in that period—that is to say, the organism breaks down the same amount of protid that it absorbs, a condition referred to as *nitrogen equilibrium*. The small amount of nitrogenous waste products which the tissue proteids furnish to the blood when they break down is balanced by the amount

of proteid taken up from the blood (circulating proteid) for the regeneration of the tissues. It is exceedingly difficult—one may say impossible—at present to follow out the change.

proteids undergo in their decomposition the end-products of their metabolism are well known, but the first katabolic changes which they undergo in the cell protoplasm we can only surmise. *Urea* is the principal nitrogenous end-product in mammals, while in birds *uric acid* takes its place. In both cases ammonia salts play an important part as forerunners of these end-products. When blood containing ammonium salts of formic, acetic, or lactic acid circulates through a fresh liver, *urea* is formed in appreciable quantity. Here, in all probability by a process of oxidation and synthesis, ammonium carbonate is formed, then, by loss of water, ammonium carbamate, and finally *urea*. It is probable that one of the most important non nitrogenous decomposition products of proteid metabolism is *sarcosulphuric acid*, which becomes distinctly increased in the blood after proteids have been taken in the food, and falls correspondingly in conditions of hunger. This acid seems to be formed in most organs, *e.g.* lungs, kidneys, etc., when blood is transfused through them, and its percentage in muscle rises during exercise. Its ammonium salt is transformed into *urea* in the liver. If oxidation processes be hindered in any way, there is an increased formation and excretion of this acid. There are many other derivatives of proteid metabolism, *e.g.* glycocholic, acetone, etc., which may appear in the urine as such or in combination with other substances (see "Urine"). The sulphur and phosphorus present in certain proteids are in large part oxidised to their full extent and excreted as simple or paired sulphates and as phosphates (see "Urine").

**Absorption and Metabolism of Carbohydrates.**—Under ordinary conditions the glucose is only taken up by the blood capillaries, but if there be a great excess of sugar in the intestine, some may also pass into the lacteals. Even after a diet comparatively rich in carbohydrates, the amount of glucose in the lymph flowing from the thoracic duct shows no increase, while the blood in the portal vein shows a rise from the normal, 0.11 per cent, to about 0.4 per cent. In order to prevent the loss of such an easily diffusible substance, the liver acts as a storehouse for the glucose, transforming it first of all into a less soluble form, glycogen, and giving it out again to the blood in the form of glucose when the percentage of the latter in the blood tends to fall below normal. By excluding carbohydrates from the diet one can gradually lower the amount of glycogen in the liver, while if one inject glucose into the blood of an animal whose liver has been rendered free from glycogen, the percentage of the latter in the liver can gradually be raised. It seems probable that these transformations of

glucose into glycogen and glycogen into glucose are the results of the activity of the liver cells, and not of the action of an enzyme produced by the latter. The muscles and many other tissues can effect this dehydration and polymerisation of glucose into glycogen as well as the liver. In fact, glycogen seems to be present at least in every living cell at some stage or other in its life-history. The muscles seem to store glycogen before the liver does, and they certainly retain it longer. Thus it is possible to exhaust the liver while the muscles still contain a fairly large quantity. In frogs the muscles can also store up glycogen even when the liver has been removed. One may say that approximately 150 g. amount of the sugar that has been absorbed by the intestine are stored up as glycogen in the liver and a slightly larger quantity in the muscles and other tissues of the body. When a larger quantity of carbohydrate is absorbed, then the liver and muscles would in all probability be unable to convert the excess into glycogen, and the percentage of glucose in the blood might rise to such a height (over 0.3 per cent) that the kidneys could no longer retain it, and the condition of glycosuria would result. But it is probable that there is another reason for temporary alimentary glycosuria in healthy individuals. In cases where large quantities of carbohydrate have been taken with a free supply of liquids, absorption may take place by way of the lacteals, and the sugar may reach the general circulation without passing through the liver. The percentage in the blood in such a case may be so high that temporary glycosuria results. When very large quantities of disaccharides, such as cane or milk sugar, are taken, some may escape inversion in the intestine and be excreted unchanged in the urine. One must remember, however, that the bowels may act as a safety-valve for the removal of the excess of sugar, diarrhoea being set up by the intestinal irritation. Proteids may also act as a source of glycogen, so that even when no carbohydrates are given in the food, a liver which has been rendered free from glycogen can store it up again if sufficient proteid be given in the food. It is important also to remember that the glycogen may accumulate in the liver from the action of substances which prevent the liver cells transforming it into glucose, *e.g.* glycerine, antipyrin, chloral, etc. Inversion of the disaccharides is necessary before they can be transformed into glycogen. The sugar circulating in the blood is normally oxidised by the tissues, furnishing finally carbonic acid and water, and in this action the muscles play an important part, as the glucose is the chief source of muscular energy. When this action of the tissues does not come into play, the percentage of glucose in the blood rises until the kidneys can no longer refrain from excreting it (see "Diabetes"). The influence of the nervous

system on the glycogenic formation and transformation in the liver is probably a vaso-motor one. Thus, after puncture of the floor of the fourth ventricle, poisoning with nitrate of amyl, curari, etc., the resultant glycosuria is due to an increased transformation of glycogen into glucose. The influence of the pancreas on the metabolism of glucose has already been referred to.

**ABSORPTION AND METABOLISM OF FATS**—This can take place to some extent even when no pancreatic juice or bile is allowed to act on the chyme, but both of these are of great importance in aiding absorption. In many cases the mixed contents of the small intestine are acid in reaction (as in dogs), and yet emulsification of the fats can take place if the pancreatic juice have free access. It is not necessary that there should be sufficient alkali present to neutralise all the fatty acids, because not only is a small amount of soluble soap able to emulsify a fairly large quantity of these acids, but in addition the salts of the bile acids can easily dissolve them and so aid absorption. Soaps and fatty acids pass partly into the blood capillaries, partly into the lymphatics, but they are transformed into fats during their passage through the intestinal mucosa, for after they have been given in the food, the lymph in the thoracic duct contains only fats.

There is no reason for believing at present that the absorption of fats takes place entirely, or even most largely, in the form of solutions of soaps, and not as an emulsion of soaps, free fatty acids, and fats. After absorption of fat, the cytoplasm of the columnar cells of the intestinal mucosa is found crowded with fat globules, while none are to be seen in the stratified hem. This is no proof, however, that the fat has not passed through in the form of an emulsion, although it has been regarded as supporting the view that the fats are absorbed in the form of solutions of the soaps, and that the latter undergo in the columnar cells synthesis with glycerine to form fats. Undoubtedly the contraction of the smooth muscular strands in the villi aids at least the passage of the chyle through the lacteals. Fats of low-melting point are more rapidly and completely absorbed than those which melt at a higher temperature. In many cases when fats foreign to the animal experimented upon are absorbed, they are deposited unaltered in the tissues, but in man the absorbed fats are not necessarily deposited in the form in which they were taken in, being usually transformed into the ordinary mixed fats of the tissues. There can be no doubt that the synthesis of fatty acids and glycerine into fats can and does take place in the columnar cells of the intestinal mucosa but, as has already been said, this does not exclude the other method of fat absorption—emulsification. Carbohydrates and proteins may also act as

sources of fat, but the proof of this cannot be given in this article (see "Diet.") The fats are deposited in various parts of the body, while a large amount, by oxidation to carbonic acid and water, sets free energy which may take the form of either work or heat. The nature of fatty degenerations and infiltrations of organs will be referred to under the special pathological headings.

**ABSORPTION OF PUTREFACTION PRODUCTS**—The products of putrefaction formed in the lower parts of the intestinal canal may also be absorbed. This is especially noticeable in cases of intestinal obstruction, when phenol, indol, skatol, etc., are absorbed, oxidised in the tissues, and excreted in combination with sulphuric acid.

**INTESTINAL MOVEMENTS**—It is exceedingly difficult to study the normal peristaltic movements of the small and large intestines, because on exposing them to the air there is a marked diminution in the peristalsis. They have been usually studied when the intestines have been surrounded by a warm saline solution. One can then perceive that peristalsis is more marked in the small than in the large intestine, and that it takes place normally in the direction towards the anus, the movements consisting of ring-like contractions passing along in the form of waves. The correct direction is maintained by the transverse folds of the intestinal mucosa acting as valves, the well-marked fold at the place of entrance of the small intestine into the caecum preventing regurgitation. Antiperistaltic movements, such as occur in reflex vomiting, are not to be seen under normal conditions. The fact that this peristalsis proceeds in one direction only, and that slowly, proves that it must be under the influence of the nervous system, and yet it persists after section of the mesenteric nerves, being also visible in separated intestinal loops. It is probable, therefore, that there are local centres present in the intestinal wall. The nerves passing to the intestine act as regulators of the movement, probably affecting these local centres. Thus stimulation of the vagus increases the rapidity or amplitude of the contractions, while splanchnic stimulation produces slowing of the peristalsis. If indifferent fluids be circulated through the vessels of a detached loop of intestine, it remains at rest, but, whenever the circulation stops, the loop shows periodic contractions. The explanation of this is that in the intestinal wall substances are formed which act as excitants to contraction, and hence their removal produces rest. The contraction of intestinal blood-vessels produced by splanchnic stimulation probably causes diminished peristalsis from the diminution in the amount of circulating blood which contains the stimulating substances. There is probably a reflex nervous mechanism between stomach and duodenum, because, shortly before the pylorus opens, the duodenum, which was previously at rest,

begins to contract. The following conditions also affect intestinal peristalsis.

1 *Alterations in temperature*—On exposing the intestines to a temperature below 7° C peristaltic movements stop, while on raising the temperature they increase. At 39° C peristalsis in the rabbit's intestine ceases, beginning again when the temperature is raised to 42° C.

2 *Effects of food and drink*—About fifteen minutes after food or after a drink of cold water peristalsis begins, while during the night the movements are markedly diminished.

3 *Effect of drugs*—Opium and atropine paralyse the action of vagus, and so hinder peristalsis, while nicotine, both after injection into a blood-vessel and into the lumen of the intestine, produces increased contractions. Potassium and sodium salts also produce contractions, the former local, the latter more general in character.

4 *Action of intestinal contents*—The bile and the intestinal gases increase peristalsis.

**FÆCES**—The faeces contain the undigested residues of food-stuffs, decomposition products, such as indol, formed in the intestine, material secreted or shed by the passages through which the food has passed, and micro-organisms of different kinds. As a rule their reaction is acid, though it may be neutral or alkaline, and their consistence also varies greatly according to the nature of the food and the time spent in the intestinal canal. The food may act upon the character of the faeces, either directly from its own composition, or from its effect upon the gland secretions which are poured into the canal, or from its action on intestinal peristalsis. Thus a vegetable diet, from the high percentage of indigestible material and of water, and from the stimulant action of the insoluble cellulose on intestinal peristalsis, produces excreta which are characterised by their fluid consistence, richness in total solids, and, as a rule, poverty in colouring material. Owing to the short period that the material is allowed to be in the canal, reduction processes do not take place to the same extent as after a flesh diet. If the vegetable diet be rich in iron, the faeces are much darker in colour. The dark colour after flesh diet is due to the presence of hematin or bodies derived from it (ferrous sulphide, etc), and also to reduced bile pigments or bodies derived from them. Under a mixed diet about 130 grammes are excreted in twenty-four hours. The most important gases present are nitrogen, hydrogen, carbonic acid, sulphuretted hydrogen, and methane. Faeces can be formed in a separated loop of intestine, and also in the intestinal canal of starving animals even when bile has not been allowed to enter the intestine. The excreta are formed in such cases from the shedding of epithelium and the subsequent decomposition of such organic material by bacteria. Different

bacteria may be found in the faeces, e.g. putrefaction and fermentation organisms and bacillus coli communis.

**DEFÆCATION**—Although the act is a reflex one, carried out through a centre in the spinal cord, it is largely influenced by the will. In man this centre is situated in the lumbal region of the cord. The pressure of the accumulated faeces in the rectum causes, by reflex action through the centre, relaxation of the sphincter, while increased intestinal peristalsis is also set up. In voluntary defaecation, the act is started by a full inspiration, closure of the glottis and fixation of the diaphragm, followed by contraction of the abdominal muscles and levator ani. The latter in this way exerts pressure on the rectum while the tension of the pelvic fascia offers the required resistance. By an effort of the will the act may, up to a certain point, be inhibited especially by increasing the contraction of the sphincter. Purgatives may act in different ways. The activity of salines depends upon their endosmotic equivalent, producing a retention of water in the intestine. Thus if salines be injected into the blood-vessels, constipation results. Some again act by increasing the amount of the intestinal secretions, while others act by increasing the rapidity or strength of the intestinal contractions (see "Constipation").

**Digital.**—Belonging to the fingers or toes or performed by the fingers, e.g. *digital nerves* (see *DEFORMITIES, Metatarsalgia, Neuritis*), *digital examinations* (see *GYNÆCOLOGY, DIAGNOSIS IN, Vaginal Examination*).

**Digitalein and Digitalin.** See DIGITALIS.

**Digitalis.** See also HEART, MYOCARDIUM AND ENDOCARDIUM (*Treatment, Medicinal*); PHARMACOLOGY, PRESCRIBING, SPASM (*Varieties, Tossu*), TOXICOLOGY (*Alkaloids and Vegetable Poisons, Foxglove*).—The dried leaves of the purple foxglove, *Digitalis purpurea*, contain the following principles—1 *Digitarin*, the most powerful, insoluble in water, soluble in alcohol. Dose— $\frac{1}{10}$ – $\frac{1}{5}$  gr. 2 *Digitalin*, the next in power, sparingly soluble in water. Dose— $\frac{1}{10}$ – $\frac{1}{5}$  gr. subcutaneously. 3 *Digitalein*, soluble in water. Dose— $\frac{1}{10}$  gr. subcutaneously. 4 *Digitonin*, not nearly so powerful, allied to saponin. 5 *Digitin*. 6 *Digitalic Acid*. 7. *Antirrhizic Acid*. Of these the first three are glucosides, and are said to represent the action of digitalis on the cardiac mechanism. The fourth is a glucoside with an antagonistic effect. The fifth is a glucoside and physiologically inert.

*Preparations*—1 Infusum Digitalis. Dose—2–4 j. 2 Tinctura Digitalis. Dose—5–15 m. The tincture contains a larger amount of digitoxin, and has therefore probably more marked

toxic and cumulative properties. Of the commercial preparations used as digitalin, *Nativelle's* consists principally of digitoxin, *Honolle's* of digitalin, and the *German* forms of digitalin.

Digitalis acts directly on the heart muscle, and also on the terminations of the vagus, causing a prolongation of diastole and a powerful increase in systolic contraction. It acts directly on the muscular walls of the arteries, causing contraction of the vessels, and also stimulates the vaso-constrictor centres. The combined effect on heart and vessels results in a marked rise of blood-pressure. Digitalis also acts on other non-striated muscles, *e.g.* the intestine, the bladder, and the uterus, but this is of less importance therapeutically. The diuretic action of digitalis is probably entirely of vascular origin. The relief of venous congestion and the general improvement in the circulation may sufficiently account for it, but many authorities believe that accompanying the general arterial contraction there is a relaxation of the renal vessels resulting in an increase of blood-flow through the kidneys. Against this latter hypothesis is the fact that it has not been shown to have a definite diuretic action in health. The chief use of digitalis is in disease of the heart. Much confusion has arisen as to the different types of cardiac disorder that are suitable for its administration, particularly as regards valvular disease. The matter is simplified if we remember that no valvular affection is in itself either an indication or a contra-indication, that it is not required when the heart is acting well and compensation good, but that, as a general rule, it should be administered, irrespective of which valve is diseased, when the pulse is weak and irregular, the arterial tension low, and evidences of backward pressure and venous congestion are present. Thus its chief sphere of usefulness is in mitral affections, but the presence of an aortic lesion is not necessarily prohibitive, for, whereas with a powerfully acting left ventricle and a strong leaping pulse its administration is not only irrational, but absolutely dangerous, its use in cases where the left ventricle is failing has frequently the most beneficial effect, particularly if large doses are given and the patient kept in the recumbent posture during treatment. In cases of combined aortic and mitral disease, or in aortic disease with secondary mitral insufficiency, digitalis acts well in combating the effects of backward pressure. Digestive disorders, if present, should, when possible, receive attention before commencing the digitalis, as they are very often aggravated by the drug. The initial action is slow, three or four days may elapse before effects on heart and kidneys are observable. It is therefore advisable to begin with large doses, say 15-20 in thrice daily, and then, when a response is obtained, to diminish the dose on account of the well-known cumulative effects. Thereafter it is only by carefully noting the

signs, symptoms, and progress of each individual case that we ascertain accurately the daily amount required. In the course of a prolonged administration excessive rapidity, slowing, or irregularity of the pulse, dyspeptic symptoms, headache, dizziness, and sudden diminution in the amount of urine passed are all indications for a smaller dose or a temporary withdrawal of the drug. At the commencement of treatment, however, such symptoms as weak and irregular cardiac action and diminished secretion of urine improve very slowly or not at all, and it is only after boldly pushing the dose for several days that we are justified in concluding that the patient is not going to react to digitalis. In fatty heart, in simple dilatation, and in some cases of cardiac irregularity and weakness without discoverable cause, digitalis may be of great service. It is also useful in a large number of acute and chronic diseases in which there is cardiac debility. In acute endocarditis it should be avoided altogether, but sometimes when the acute stage is over and the patient is convalescent small doses give favourable results. It must be given with great caution when the arteries are degenerated, on account of the risk of cerebral hemorrhage. In uncomplicated Bright's disease it is very rarely indicated, certainly never as a diuretic. In rare cases, however, both in the acute and chronic forms, weak and failing heart action constitutes the most serious symptom, and may of itself call for the use of digitalis. In hemorrhage digitalis does more harm than good, the arteriole contraction being more than counterbalanced by the increased pulse tension and force of the heart's action.

**Diglossia.**—A rare malformation, in which there is duplicity of the tongue, usually due to fissure of the organ (*Schistoglossa*).

**Dihydric Alcohols.**—The dihydric or diatomic alcohols are dihydroxyl-derivatives of hydrocarbons, two hydrogen atoms being displaced by two hydroxyl groups, thus from benzene ( $C_6H_6$ ) is got resorcin ( $C_6H_4(OH)_2$ ).

**Dilatation.**—Enlargement or expansion, or the part enlarged or expanded. Thus there is dilatation of the heart, of the bronchial tubes (bronchiectasis), of the stomach, of the veins (varix), of the arteries (aneurysm), of the cervix uteri (in labour), and of the cerebral ventricles (hydrocephalus).

**Dilator.**—An instrument used for the purpose of opening up or expanding a natural opening, passage, or canal, *e.g.* Holt's urethral dilator, Hegar's or Barnes's cervical dilators, and Bossi's metal dilator used in accouchement forcé, also a muscle which causes dilatation of an opening, such as the dilator pupillæ and the dilatator narium.



**Dilecanus.**—United twins or double monster, in which the head and chest are single and the pelvis double (Gr *lêkos*, a dish or pelvis), the term was introduced by Taruffi (1889), and the pelvis may be situated side by side (*Dilecanus dipleurus*), or may be attached to each other in the pubic regions (*Dilecanus bispagus*).

**Dill Fruit.** See ANETHI FRUCTUS

**Diluents.**—Medicines or indifferent substances which increase the proportion of water in the blood and other bodily fluids, which, as it is said, "thin the blood." Water is the most commonly employed, and its action is aided by the addition to it of lemon peel or an acid (which increases the flow of saliva). Diluents render the urine less irritating, diminish thirst, and remove products of disordered metabolism from the body, they are therefore used in cystitis, diabetes, fever, gout, rheumatism. See PHARMACOLOGY, PRESCRIBING, etc

**Dimethylamine.**—A liquid ( $\text{NC}_2\text{H}_7$ ) having an ammoniacal smell, it has been found in decomposing fish, and is a ptomaine or animal alkaloid. Dimethyl ( $\text{C}_2\text{H}_5$ ) is ethane or ethyl hydride, in dimethylamine two of the hydrogen atoms of ammonia have been replaced by ethane. See SNAKE-BITES AND POISONOUS FISHES (*Poisonous Fish, Putrefaction*)

**Dimethylarsine.** See CADOTYL

**Dimethylbenzene.** See XYLENE

**Dimidiate.**—Divided into two, double, e.g. a placenta consisting of two nearly equal parts is a dimidiate or duplex placenta

**Dimple, Postanal.**—A depression or umbilication found immediately behind the anus, over the lower end of the sacrum or coccyx, foveola coccygea, it may be surrounded by a ring of hair, it may be associated with spina bifida, and may be the seat of a fistula, or sinus, or cyst, or dermoid. See Ballantyne's *Antenatal Pathology*, vol. II p. 327

**Dinitrobenzine.** See TRADES, DANGEROUS (*Aniline Poisoning*)

**Dinomania.**—Choreomania or dancing mania (from Gr *divos*, a whirl, and *mania*, madness).

**Dinsdale-on-Tees.** See BALNEOLOGY (*Great Britain, Sulphur*)

**Diectophyme Gigas.**—*Strongylus gigas*, a large nematode occasionally found in man. See PARASITES (*Nematodes, Strongylida, Eustrongylus gigas*)

**Diodon.**—Porcupine fish, regarded as poisonous. See SNAKE-BITES AND POISONOUS FISHES.

**Diodoncephalus.**—The deformity in which there is a double row of teeth in the same jaw

**Dioestrus.**—The short resting stage between two oestrous cycles as seen in polyoestrous mammals, such as the sheep and the mare. See ANÆSTRUM.

**Dionin.**—Dionin is ethylmorphine hydrochloride ( $\text{C}_{19}\text{H}_{23}\text{NO}_3 \cdot \text{HCl} \cdot \text{H}_2\text{O}$ ), and has the medicinal properties of morphine, with, it is said, less liability to produce the morphine habit, it has been specially recommended (in doses of  $\frac{1}{4}$  to  $\frac{1}{2}$  grain) in whooping-cough, in asthma, in eye practice, and in internal painful conditions (gastric cancer or ulcer, colic, etc.).

**Dioptre.**—The unit of optical power, the optical strength or refractive power of a lens of one metre focal length ("A convex lens interposed in the path of a parallel beam of light brings the light to a focus at a constant distance from itself, which is called the focal length of the lens") See REFRACTION (*Lenses*) The dioptric mechanism by which an object is seen as single by two eyes is described in the article PHYSIOLOGY, THE SENSES (*Vision, Monocular*)

**Dioxybenzene.**—Diphenol, benrene in which 2 atoms of hydrogen have been replaced by 2 molecules of hydroxyl,  $\text{C}_6\text{H}_4(\text{OH})_2$ , there are three isomeric varieties—(1) orthodioxycbenzene or pyrocatechin, (2) metadioxycbenzene or resorcin, and (3) para-dioxycbenzene or hydroquinone. See PHYSIOLOGY, EXCRETION (*Urine*)

**Dioxypurin.**—Xanthin ( $\text{C}_5\text{H}_4\text{N}_4\text{O}_2$ ).

**Diphallus.**—The deformity (probably a low degree of double monster) in which the penis is double, there may be evidence of duplication of the lower end of the spinal column in such cases as well as of the bladder

## Diphtheria.

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See also ALCOHOL (*Indications in Diseases of Childhood*), AMBLYOPIA (*Torae, after Diphtheria*), ASPHYXIA (*Causes*), BRAIN, INFLAMMATIONS (*Acute Encephalitis, Etiology*), BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Cerebral Thrombosis, Causing Hemiplegia after Diphtheria*), DISINFECTION (*Incubation Periods and Quarantine*), GASTRO-INTESTINAL DISORDERS OF INFANCY (*Diseases of the Oesophagus, Diphtheria*), HAMATEMESIS (*Levons*), HEART, MYOCARDIUM AND ENDOCARDIUM (*Simple Endocarditis*), IMMUNITY (*Diphtheria*), INFECTION (*Rules for Prevention of Infectious Diseases*), INTUBATION, JOINTS, DISEASES OF (*Pyogenic Diseases*), LARYNX, NEUROSES OF (*Paralysis of Vocal Cords*), LEUCOCYTOSIS (*Inflammatory and Infective Conditions*), MEASLES (*Diagnosis*), METEOROLOGY (*Seasonal Prevalence of Diseases*), NERVES, NEURITIS (*Causes of Mono-Neuritis*), NERVE, MULTIPLE PERIPHERAL NEURITIS (*Diphtheritic*), OCULAR MUSCLES, AFFECTIONS OF (*Etiology*), OXYGEN (*Inhalation and Hydrogen Peroxide Application*), PNEUMONIA (*Pneumococcus*), PUPERIUM, PATHOLOGY (*Septicæmia, Diagnosis*), PUPERIUM, PATHOLOGY (*Nipples, Diphtheria*), PURPURA (*Symptomatic, Toxin*), STOMACH AND DUODENUM, DISEASES OF (*Membranous Gastritis*), THERAPEUTICS, SERUM-THERAPY (*Antidiphtheritic Serum*), TRACHEA, AFFECTIONS OF (*Tracheotomy*).

DIPHTHERIA is an inflammatory infectious disease, usually of mucous membranes, of which the essential cause is a specific micro-organism. The inflammatory process is often attended by the formation of false membranes. The absorption of the toxic products of the micro-organism produces secondary effects, of these a peculiar form of paralysis is the most characteristic.

The name *diphtheria* we owe to Bretonneau (1855). He had previously (1821) used the term *diphtheritis*.

#### HISTORY AND GEOGRAPHICAL DISTRIBUTION.

The disease was first described by Aretæus of Cappadocia in the second century A.D., who mentions that it was prevalent in Egypt and Syria. Aretæus, in the fifth century, was acquainted with it. During several succeeding centuries there occur at long intervals of time only a few doubtful records of the disease, and it is not till the sixteenth century that a more definite account is forthcoming. In 1517 Hecker described a fatal form of infectious angina that appeared in Holland, and later in Basle. During the latter half of the century the disease was prevalent in Southern Italy, Spain, and Portugal, there were also outbreaks in Paris and Dantzic. Epidemics continued to occur during the seventeenth century in Italy, Spain, and Portugal, and in the following century Northern France, Holland, Switzerland, Germany, Sweden, England, and North America were invaded. Towards the end of the eighteenth

century, however, diphtheria seems to have been somewhat diminishing in its prevalence in the countries we have named, and though from time to time outbreaks are reported in various quarters during the first half of the nineteenth century, it was not till its middle that the disease began again to be serious over a wide area. Since then it has been and still is, more or less, prevalent all over Europe and in North America and Australia, to a less extent also in certain districts of South America, South Africa, India, and China.

**ETIOLOGY.**—At the present day, therefore, diphtheria is a very widely spread affection. The accumulation of properly compiled records of the fatal cases in various countries during the past twenty-five to fifty years has enabled inquirers to elucidate several important facts with respect to its etiology. Amongst the most recent and valuable researches in this field are those of Newsholme. This author draws conclusions, of which the following is a brief summary—

Diphtheria has a tendency to spread from one place to another by the ordinary channels of communication. In certain years diphtheria may be pandemic over whole countries or a continent. The amount of endemic diphtheria varies greatly for different countries and cities, but "in no town from which records have been obtained is there a complete absence of the disease in a single year since the records commenced." In places where the amount of endemic diphtheria is not great, epidemics tend to occur in cycles, the intervals between the cycles being very variable for different places. The duration of an epidemic is also variable, but is usually longer in large than in small cities or towns.

Before the appearance of Newsholme's work it had been shown that for England and Wales one of the most striking features with respect to the prevalence of diphtheria was that, whereas up to 1880 the disease was incident upon the rural to a greater extent than upon the urban population, since that date the reverse has been the case, there has been an increase both in rural and urban diphtheria, but the urban incidence has risen to a much higher degree than the rural. London has especially suffered. Newsholme's observations show that this increase in urban diphtheria is not confined to England and Wales, but has also occurred in countries so widely separated as the United States, Japan, and South Australia. It is reasonable to suppose that the wonderful improvements effected during recent years in our means of transit have had no small share in contributing to this increase.

Like most infectious diseases, diphtheria has its special *seasonable prevalence*, which, when estimated by the recorded deaths, is in this country from September to the end of the year. In London a marked rise in the notifications is observed in July. The consideration of the *influence of soil and climatic conditions* upon the

prevalence of diphtheria has led to considerable diversity of opinion. While some writers deny that these conditions exert any material effect, it is held by others that a soil which is continually moist and impregnated with organic refuse is favourable to both the existence and violence of the disease. Newsholme, in the work from which we have already quoted, gives the results of his inquiries into the relation between epidemic diphtheria, the rainfall, and the level of the ground-water. The general conclusions to which he comes are abbreviated by himself as follows:—

"An epidemic of diphtheria never originates when there has been a series of years in which each year's rainfall is above the average amount. An epidemic of diphtheria never originates or continues in a wet year (*i.e.* a year in which the total annual rainfall is materially above the average amount), unless this wet year follows on two or more dry years immediately preceding it. The epidemics of diphtheria, for which accurate data are available, have all originated in dry years (*i.e.* years in which the total annual rainfall is materially below the average amount). The greatest and most extensive epidemics of diphtheria have occurred when there have been four or five consecutive dry years, the epidemic sometimes starting near the beginning of this series, at other times not until near its end. Dry years imply low ground-water, and we find, therefore, in the years of epidemic diphtheria that the ground-water is exceptionally low. The exact variations in the ground-water which most favour epidemic diphtheria cannot with the data to hand as yet be stated, but it is probable that when this is cleared up, it will become clear why, in exceptional years which have a deficient rainfall, epidemic diphtheria is either absent or but slight."

Newsholme's conclusions are at variance with the hitherto accepted ideas concerning the association of dampness of soil and diphtheria, but they are based on accurate records. Taking up an idea put forward by M. A. Adams, of Maidstone, Newsholme suggests that the micro-organism of diphtheria passes a saprophytic existence in the soil, and that its growth is favoured by a low level of the ground-water, such as would follow a deficient rainfall. The rise of the ground-water consequent upon rains drives the micro-organism out of the soil, the organism may then become parasitic on man, and an epidemic arise. But this hypothesis, which may account for the autumnal and winter prevalence of diphtheria in England, and for epidemics which arise in wet following dry years, does not appear to explain the epidemics which commenced in dry years, unless it be that the rains that do fall during the dry years are sufficient for the purpose suggested, or that variations in the atmospheric pressure, apart from rainfall, have the same effect. Moreover, the diphtheria bacillus has not yet been found

in the earth. Diphtheria does not appear to flourish in tropical to such an extent as in temperate climates.

There is evidence to show that in some epidemics of diphtheria the disease increases in virulence as the epidemic progresses. At first sore throats of a mild character and questionable nature occur, then in increasing numbers cases present themselves that are undoubted and more severe, even proving fatal, so that it seems as if the organism of the disease gains in virulence as it is transmitted from one person to another.

It is believed by some that throat lesions (*e.g.* simple tonsillitis or the angina of scarlet fever) predispose to diphtheria. But the writer ventures to doubt whether a throat affection renders the individual more susceptible than does any disease which lowers the power of resisting the attacks of pathogenic micro-organisms.

*Association with other Diseases.*—Diphtheria is frequently found associated with other zymotic diseases. It appears to have an especial liking for scarlet fever and measles, but it not infrequently attacks patients suffering from pertussis, varicella, and tuberculosis, less commonly it is found with enteric fever and small-pox. It complicates both the acute and convalescent stages of these affections, and is more fatal when thus combined than when occurring alone. The occurrence of these coincident infections is largely determined by such factors as the age-incidence and prevalence of the diseases concerned. Thus we find from the published statistics of the Metropolitan Asylums Board that scarlet fever attacks the patients convalescing from diphtheria quite as often as diphtheria does those recovering from scarlet fever.

*Age and Sex and Case-Mortality.*—The London notifications for 1892 to 1897 show that rather more than one-third of the cases are under the age of five years, and rather less than one-third are between the ages five and ten. With every succeeding quinquennium the number becomes very much smaller. Age has also a marked influence on the case-mortality. This is highest in infants under one year. It then gradually falls up to the fifth year, and after that more rapidly, but it rises again after forty. Before the introduction of the antitoxin treatment the case-mortality of the patients under five admitted to the hospitals of the Metropolitan Asylums Board was 50 per cent, from five to ten, 28 per cent, from ten to fifteen, 10 per cent, from fifteen to twenty, 4 per cent, from twenty to forty, nearly 5 per cent, and over forty, 17 per cent.

Diphtheria attacks more females than males, this is usually attributed to the fact that the nature of the duties and habits of the female sex render its members more exposed to infection than the male.

*Modes of Dissemination.*—By far the most common mode is by personal communication

between the affected and the healthy, either directly as in such an act as kissing, or perhaps more often indirectly by means of utensils for eating and drinking, handkerchiefs, toys, etc. The infection may be harboured for a considerable time in such articles as wearing apparel and toys. It is not often conveyed by third persons.

There are now on record several epidemics of diphtheria in which it has been clearly proved that the infection was conveyed in *milk*. In some instances the source of infection has not been traced, in others the specific contamination has been derived from a human source during the collection and distribution of the milk, in a third group the infected milk has been obtained from cows that have been at the time the subjects of a disease of the udder, a febrile affection in which the local lesions consist of vesicles which pass on to pustules and ulcers. It was suggested by Power that the connection between this disease and the specific infection of the milk was more than accidental. Working at the subject experimentally, Klein found that in five out of ten milk cows inoculated in the shoulder with a virulent growth of diphtheria bacilli, there was produced after a few days an eruption on the teats and udders similar to that occurring naturally. The disease could be transferred to calves by inoculation. In two out of the five cases diphtheria bacilli were obtained from the milk yielded by the animal, and in two the bacilli were shown to be present in the vesicles and pustules upon the udder. The experiments of Klein, so far as they go, corroborate Power's hypothesis. It is true that the disease set up by Klein in the cow differs considerably from that met with in the human being, notably in the production of vesicles and pustules containing the specific organism in a region remote from the seat of inoculation. With respect to the two similar experiments on cows by Abbott (of Philadelphia) with negative results, it may be observed, firstly, that some of Klein's own experiments were also negative, and that the negative can hardly be allowed to weigh against the positive results, secondly, that Abbott did not observe the same conditions as those under which Klein worked. The specific bacillus has been found in milk presumed to be the cause of an outbreak of diphtheria (Bowhill and Eyre), but it has not yet been demonstrated in the milk and in the lesions on the udder of a cow to which a definite epidemic has been traced.

Besides its probable occurrence in cows, diphtheria certainly attacks cats, and instances have been recorded both where it has been communicated from the animal to man and from man to the animal.

There is no instance of diphtheria having been conveyed by the water-supply, nor do faulty sanitary arrangements in connection with

the removal of sewage or refuse lead to outbreaks. There has been marked improvement made in the sanitary condition of this country during the past thirty years or so, which has resulted in a notable lessening in the prevalence of enteric fever, a disease which certainly goes with bad sanitation of the kind to which we have alluded, yet diphtheria has become more rife during that period. But there is reason to believe that insanitary conditions may aggravate or possibly predispose to diphtheria, and in this way, therefore, there may be said to be a connection between the disease and its surroundings.

*School Influence*.—When we remember that diphtheria is especially a disease of children, we might argue *a priori* that the daily aggregation of numbers of such individuals would tend to widen its prevalence. And as a matter of fact it has been shown by more than one observer (especially Thorne Thorne and Power) that a local prevalence of the disease has been kept up by such aggregation. Not only is it that children catch the disease by being brought into contact with infected children at school, but they go home and infect others who are not attending school. In some instances the closing of the school has markedly diminished the prevalence of diphtheria, and the reopening has increased it. In certain epidemics occurring in not very populous rural districts the school influence has not been difficult to trace, and cannot be gainsaid. But the question arises whether or not this influence is in operation over larger areas and for longer periods.

It is not claimed by those who are most conversant with the problem that the marked increase of diphtheria in this country during recent years is entirely due to school influence, but it is suggested that a considerable portion of it is. The two facts that are brought forward in favour of this view are as follows.—The Education Act was passed in 1870, and since that date there has been from time to time further legislation, which, while it has afforded greater facilities to those who are willing to send their children to the Board Schools, has brought more compulsion to bear upon those who are not. The age of compulsory attendance is from three to fifteen years. Making allowance for changes due to other causes that can be fairly well ascertained, there has been since 1870 in London and in the provinces (including Wales) a greater increase in the mortality (per million living) at the age-period three to fifteen years than was to be expected, comparison being made with the age-periods under three and over fifteen, and with the years immediately preceding 1870. The other fact is that since the compulsory notification of diseases was introduced into London there has been a marked fall in the already rising notifications at the age-period three to fifteen soon after the commencement of the summer holiday (four weeks) of the School.

Board, the fall continuing till shortly after the reopening of the schools at the end of the holidays, when the notifications rise again. But the question is not absolutely settled. According to Newsholme, in certain of the large continental cities where compulsory school attendance has been in force for many years, it seems to have had no great influence one way or the other upon the prevalence of diphtheria. Newsholme, however, is dealing with the mortality of diphtheria for all ages. What is required is a comparison of the mortality for the school ages with that for other ages. According to Murphy, the figures for Berlin, as far as they go, tell the same story as those for London.

**CLINICAL HISTORY.**—The *incubation period* is from twelve hours to four days.

Though any of the mucous membranes may be the seat of the disease, yet in the vast majority of cases the fauces alone are affected. Next to the faucial the nasal and laryngeal forms are the most common. The conjunctiva, genital organs, and cutaneous wounds are rarely affected, still more rarely the œsophagus, ear, stomach, urethra, and bladder.

*Faucial diphtheria* is very insidious in its onset. Such striking symptoms as frequent vomiting or a rigor are rare. The patient, usually a child, is observed to look ill and refuses his food. The cervical glands may be enlarged, and the parents perhaps think the child has mumps. If on this sign of illness the temperature be taken, it will be found to be raised ( $101^{\circ}$  to  $105^{\circ}$  F). The pulse-rate is slightly accelerated. On inspection of the fauces the tonsils are seen to be slightly swollen and covered with a glazy film. In adults, and sometimes in children, sore throat is the first symptom. The subsequent course of the attack varies. In a fatal case, untreated or treated too late with antitoxin, it is as follows.—The glazy film becomes a definite yellowish exudation of a distinctly membranous character. It spreads from the tonsils over the soft palate, uvula, and pharynx (very rarely to the mouth and tongue), often forming a continuous sheet. The mucous membrane beneath is inflamed and swollen, though only occasionally to an extreme degree, and from it a slight oozing of blood takes place. After five or six days the exudation begins to decompose and separate. It turns a greyish black colour, and from it a peculiar and very offensive odour is given off. After separation there is often repeated re-formation of membrane. In some cases the exudation may have quite disappeared before the fatal issue, which usually takes place in seven to fourteen days from the onset. Meanwhile, with the extension of the local affection, the lymphatic glands below and behind the jaw become moderately enlarged and painful. Occasionally also the skin and subcutaneous tissue of the neck are swollen. The nasal passages are frequently invaded, and a

blood-stained watery discharge constantly trickles from the nostrils, and from time to time membranous casts of the nasal fossæ are expelled. The temperature falls quickly after the initial rise. Its subsequent course is very irregular. Briefly it may be stated that a fresh extension of faucial exudation, or the occurrence of adenitis, cellulitis, or broncho-pneumonia, leads to a rise of temperature, but that during the last three or four days, after the patient has fallen into the final state of prostration, it is subnormal. The pulse during the first few days is moderately frequent (100 to 130) and compressible. Then with the increasing heart-failure it falls in rate, and the tension becomes lower. In most cases there is albuminuria. The constitutional symptoms are those due to progressive cardiac failure, increasing pallor and prostration, diminished flow of urine, and frequent vomiting. Towards the end the patient is drowsy, but restless. The mind is quite clear and delirium is exceptional. Death is due to syncope, and is often attended by a slight convulsion.

In non fatal cases the symptoms differ only in degree from those just described. As a rule the severity of the constitutional symptoms depends upon the extent of the local lesion, which varies greatly. In the very mildest forms there will be two or three small patches of exudation limited to one or both tonsils. In cases not so slight there are numerous patches on the tonsils, uvula, and palate. The exudation is not necessarily membranous, but may be cheesy, pulsatious, or gelatinous. It may commence at one spot or simultaneously at many. But the favourite place of origin is one of the tonsils. Occasionally it begins in the tonsillar crypts. In mild cases it remains limited, and lasts but a short time. In such cases there may be no constitutional symptoms.

*Nasal diphtheria* is most commonly found accompanying the faucial and laryngeal varieties. But the disease may remain limited to the nasal passages. There is a discharge, at first watery, afterwards mucopurulent, and occasionally blood-stained, often it is offensive and excoriates the nostrils and lip. The nose is more or less blocked, and casts of the nasal passages may be shed. The constitutional symptoms are rarely severe. The discharge is often chronic.

*Laryngeal diphtheria* is in the large majority of cases secondary to the faucial form. It occurs in from 11 to 15 per cent of all cases of clinical diphtheria, and arises within a week from the onset of the primary affection. It is quite the exception for diphtheria to start in the larynx. But inasmuch as the larynx may, and often does, become involved in very slight cases of the faucial disease, the symptoms due to its invasion may be the first signs of the illness of the patient. The symptoms are those of laryngeal obstruction, aphonia, stridor, a frequent harsh cough, and recession of the chest-walls.

Any one of these symptoms may occur earlier, or be more prominent than the others. At intervals of a few hours there are in some cases attacks of urgent dyspnoea, in which the patient becomes much distressed and cyanosed. These attacks, which arise and pass off suddenly, are usually due to spasm of the laryngeal sphincter. They may, however, be due to temporary blocking of the larynx by a piece of loose membrane. If the diphtherial process spreads to the trachea and bronchial tubes there is advancing and permanent dyspnoea and lividity, and the patient dies of suffocation. On the other hand, it is not at all infrequent, even amongst cases not treated with antitoxin, for the progress of the disease to be arrested at the larynx, and the patient to recover without any urgent symptoms of laryngeal obstruction. In a few cases casts of the larynx, trachea, and bronchia may be coughed up. Lastly, it must be remembered that the larynx and respiratory tract below may be invaded without there being any symptoms to point to such an event. This masked form of the disease is especially met with in very severe cases of faucial diphtheria, the laryngeal being obscured by the extreme severity of the other symptoms. But it may also be observed in less severe cases amongst adult patients. In them the width of the respiratory tract allows of the formation of membrane without interference with respiration, and it is not until the bronchioles become blocked that dyspnoea arises. In these cases, however, there is usually aphonia, which is therefore an extremely grave symptom in diphtheria of adults.

In rare cases there is expectoration of casts of the trachea and bronchi, with absence of any such signs as characteristic obstruction of the larynx or the respiratory tract below it.

In diphtheria of the *conjunctiva*, membrane forms upon the palpebral, rarely upon the ocular conjunctiva. The eyelids are swollen. The cornea may become inflamed and ulcerated, perforation may result, leading to pan-ophthalmitis (*vide* "Conjunctiva"). In *nodal diphtheria* the labia minora and the inner surfaces of the labia majora are covered with ashy grey membrane, which may spread into the vagina. There is intense inflammation of the external genitalia and swelling of the neighbouring lymphatic glands. The constitutional symptoms are severe, and are similar to those present in the faucial form.

Diphtheria of other mucous membranes is rare.

*Wound diphtheria* occurs in two forms. In one the membranous exudation appears upon a previously existing wound, in the other the wound is caused by accidental inoculation with the diphtheria bacillus. A small chronic abscess is the result.

In severe cases of diphtheria, especially of the faucial variety, the local exudation may persist

for three or four weeks. In rare cases of the faucial, nasal, and laryngeal forms the local affection may be of much longer duration, two to six months. Such cases are termed *prolonged diphtheria*. They commence in the same manner as do the ordinary cases. The larynx may become involved after the faucial affection has lasted some weeks. Except in the nasal form a fatal termination is common.

**ASSOCIATED SYMPTOMS.**—A few words are required concerning certain symptoms and conditions more or less common in the severer forms of diphtheria. *Albuminuria* is found in from 50 to 75 per cent of the cases. It may appear at any time during the first three weeks, sometimes later. Most commonly it is observed about the fourth day. Its duration and amount are very variable. Its presence is of importance as roughly indicating the extent to which the absorption of the diphtherial toxins has been carried. The more persistent albuminuria, and the greater the quantity of albumin, the more severe is the case, and the more likely is paralysis or cardiac irregularity to follow. Except in one other particular, the condition of the urine does not deviate from the normal. We refer to the diminution of the daily quantity. In most severe cases this occurs to a certain extent, but there may be complete *suppression of urine*. Usually this condition comes on about the sixth or seventh day. With it there are frequent vomiting, alidity, and cardiac failure. Death occurs after two to four days of total suppression. Occasionally fatal suppression arises in patients who appear to be recovering from the attack of diphtheria.

*Hæmorrhagic diphtheria*.—In some severe cases after a few days' illness cutaneous and subcutaneous hæmorrhages appear, together with a constant oozing of blood from the nose, pharynx, and gums, hæmatemesis and melæna. These cases are almost invariably fatal. Hæmorrhages are found post-mortem in the retro-pharyngeal and peritoneal tissues, beneath the pleura and mucous membrane of the stomach, and into the lungs, cardiac and voluntary muscles. In rare cases a purpuric condition arises during the period of convalescence.

During the acute stage of diphtheria there is a marked increase in the number of leucocytes in the blood. This condition disappears during convalescence.

**COMPLICATIONS.**—With the exception of paralysis and certain cardiac complications, these usually arise during the period of the local affection. The most common are *otitis media* (in about 7 per cent of all the cases), *lobular pneumonia* (3 per cent, especially in laryngeal diphtheria), and *cervical adenitis, cellulitis, and abscess*. *Lobar pneumonia, nephritis, convulsions, endocarditis, venous and cardiac thrombosis, embolism, pyæmia, and erythematous rashes* are rarely met with. Though the local

exudation usually clears off without leaving any loss of tissue, yet occasionally *sloughing* and *ulceration* result, and may lead to *septicæmia*.

The most important and striking complication, or more frequently sequel, of diphtheria is *paralysis*. The proportion of cases in which it occurs varies. At the Eastern Hospital during 1892 and 1893, before the introduction of the antitoxin treatment, it developed in about 18 per cent of those patients who survived the acute stage of the disease, and in 12 per cent of all cases. It occurs more frequently, relatively as well as absolutely, in children than in adults, it is more often observed to follow a severe than a mild attack of diphtheria, and though it may succeed any form of the disease, yet it is met with more frequently after the faucial variety.

Paralysis usually sets in during the second, third, or fourth week from the commencement of the attack of diphtheria, but it may arise as early as the fifth day, or as late as the twelfth week. The first symptom is commonly a nasal voice, or a regurgitation of liquid through the nose during the act of drinking. The soft palate will be found to be motionless, and its sensation and reflex impaired or lost. Often the paralysis remains limited to the palate, but it may progress to a greater or less extent, various muscles or groups of muscles becoming affected in a sequence more or less regular. The ciliary muscles are involved early, and the patient is unable to define near objects clearly. Then the gut becomes unsteady and the lower extremities weak. Strabismus and other signs of ocular paralysis are observed, and, lastly, the muscles of the neck, trunk, upper extremities, and respiration being affected, the paralysis becomes generalised, and the patient is helpless.

The frequency with which the different groups of muscles are involved may be gathered from the following figures.—Of 125 consecutive cases of paralysis analysed by the writer (see *Brain*, 1895), in 102 the palate was affected, in 56 the ciliary muscles, in 52 the lower extremities, in 26 the external ocular muscles, in 21 the upper extremities, in 14 the larynx, and in 10 the diaphragm. In 19 cases difficulty in swallowing was present, but this number is probably too small, because many patients are placed on nasal feeding at an early stage of the disease, so that paralysis of the pharyngeal muscles occurring subsequently escapes notice. It is rare for the sphincters of the bladder or rectum to be involved. Occasionally there is weakness of the oral muscles, but marked facial paralysis is exceptional. Paralysis of the tongue is very rare.

Frequently the paralysis remains limited, this was the case in 66 of the 125 cases (52.8 per cent). In 28 it was confined to the palate, in 17 to the ciliary muscles, in 11 to the palate and ciliary muscles, in 6 to the palate and legs,

in 3 to the palate and ocular muscles, and in 1 to the respiratory muscles.

Of the extrinsic muscles of the eye the external recti (usually the left) are most often affected. Complete ophthalmoplegia is rare. The reactions of the pupils become sluggish, but are seldom abolished. In laryngeal paralysis most commonly all the muscles are involved, so that there is aphonia and a toneless, ineffectual cough. Abductor paralysis is exceptional.

The loss of muscular function is rarely complete, even in the most severe and generalised cases. There is paresis rather than paralysis. Sensation may also be impaired. Adults will complain of tingling or numbness of the tongue, fingers, and toes. Careful testing will then usually elicit impairment of common sensation. The special senses are very rarely affected. Optic neuritis and atrophy do not occur. The knee-jerks are as a rule abolished. Indeed it is not uncommon to find them absent in cases of diphtheria before paralysis has set in, and even in cases where there is never paralysis at any time. It may be months before they are restored. Occasionally a stage of exaggeration precedes their disappearance. The superficial reflexes remain in all but the most severe cases. In the generalised form there is much wasting. The electrical reactions are diminished, but the reaction of degeneration is uncommon.

Disturbance of the circulation was met with in 29 of the 125 cases. The heart's action is increased in frequency and is irregular. Occasionally there are more severe symptoms such as are mentioned below under cardiac complications.

In 4 cases, in all of which there was paresis of the respiratory muscles, there was respiratory irregularity of the nature of "Cheyne-Stokes breathing." In rare instances sudden attacks of dyspnoea occur (respiratory crises).

Thirteen of the 125 cases (10.4 per cent) died of paralysis or its effects, 6 of these were fatal from cardiac failure, 4 from respiratory paralysis, 2 from persistent vomiting and cardiac failure, and 1 from convulsions. When there is respiratory paralysis there is great risk of suffocation from accumulation of mucus in the lungs. In paralysis of the pharynx and larynx the patient may be choked by entrance of food into the larynx if care in feeding be not exercised.

In severe generalised cases the patient becomes very apathetic, and appears not to notice what is taking place around him. The saliva accumulates and dribbles from the mouth or, unless the head be lowered, trickles into the larynx. Muscular twitches, especially of the face, are sometimes observed.

The duration of an attack of paralysis varies from a few days to ten or twelve weeks, chiefly according to the extent of the paralysis. When

once the patient begins to improve, recovery, which is always complete, progresses quickly. There does not appear to be any relation between the severity of the paralysis and the interval of time that elapses between the attack of diphtheria and the onset of paralysis. But in generalised cases the more rapidly the paralysis spreads the more likely is the issue to be fatal.

The introduction of the antitoxin treatment has had a marked effect upon the incidence of paralysis. On the whole this has been increased, probably because a larger number of severe cases are tidied over the acute stage of the attack of diphtheria. Indeed, this increased incidence has taken place entirely in those cases brought under the treatment at a late stage. Amongst cases treated early the incidence has diminished very considerably. According to Woollacott (*Lancet*, 26th August 1899) large doses of antitoxin (4000 units and upwards) are probably more effective than small in preventing paralysis and lowering its mortality.

The much and rightly dreaded *cardiac complications* are usually met with from the end of the first to the fifth or sixth week, but in paralytic cases they may arise later. The most common is *dilatation*, which leads to irregularity and attacks of syncope. When acute, the dilatation is accompanied by vomiting and severe epigastric pain. Irregular rhythm, tachy- and bradycardia may also occur with, but also independently of, dilatation. All these conditions are very grave, apart from a fatal issue they are apt to persist for a considerable time in spite of careful treatment.

*Anæmia* is a frequent sequel of an attack of diphtheria.

A marked *relapse* occurs in about 1 per cent of the cases. As a rule it is not so severe as the primary attack. Secondary attacks of sore throat, not clinically diphtheria, are still more common. Probably they are of the nature of relapses. *Second attacks*, occurring after several months or years, are not uncommon. It is probable that an attack of diphtheria confers little, if any, lasting *protection* against another attack.

**MORBID ANATOMY AND PATHOLOGY** — The essential cause of diphtheria is a bacillus, often called, after its discoverer, the Klebs-Loeffler bacillus. This organism, either itself exciting an inflammation of a mucous surface or cutaneous wound, or, less frequently, becoming active in an inflammation previously set up by some other agent, gives rise to an exudation which in the majority of cases is distinctly membranous.

**Membrane** — To some mucous surfaces, especially that of the fauces, this membrane is very closely adherent. Consequently, it is with difficulty separated therefrom, and its removal is often attended with bleeding. But to the mucous surface of the nasal fossæ, larynx, trachea, and

bronchi, it is as a rule much less closely attached. When the membrane has separated naturally, breaches of the underlying structure are left which are usually slight, though occasionally they are both deep and extensive. Microscopically the false membrane consists of a hyaline fibrinous matrix, having either a granular or fibrillated appearance, in which a leucocyte can here and there be seen. It may also be distinctly laminated. When it is closely adherent to the mucous membrane, the latter is more or less deeply infiltrated with fibrin and leucocytes, and its epithelium is necrosed and incorporated with the false membrane. Beneath the infiltrated layer the tissues exhibit the appearances of inflammation. *Micro-organisms present* — The diphtheria bacilli are found upon the surface of the false membrane, usually in a continuous layer. Occasionally they penetrate as deeply as the mucous membrane. Other micro-organisms are also present, especially strepto- and staphylococci, and they may be found invading the adjoining mucous membrane. Exceptionally the Klebs-Loeffler bacilli are found in the lymphatic glands, spleen, lungs, and other organs, and also in the blood. Probably this general invasion of the body takes place shortly before death, when all resistance to the entrance of the bacilli has been overcome.

*Gross pathological changes*. — In most cases an autopsy reveals to the unaided eye little beyond the local lesions. The skin, heart-muscle, and kidneys may be unusually pale. The cervical and occasionally other lymphatic glands are inflamed. Microscopically the heart-muscle is found to have undergone a fatty degeneration more or less extensive. Sometimes also there is a similar change in the renal epithelium, but the lesions of nephritis are rare. In cases fatal during paralysis there is degeneration of the affected nerves. These changes are found mostly in the peripheral nerves. There is a breaking up and disappearance of the white substance of Schwann, followed by a degeneration of the axis cylinder.

**The bacillus** — The bacillus of diphtheria is a rod-shaped, non-motile, polymorphous organism. It varies in length from about 1.5 to 6  $\mu$ . Often its protoplasm does not stain uniformly, and hence the bacilli have a beaded appearance. The longer forms are frequently clubbed at one end, and slightly curved. The shorter forms may be wedge-shaped. Usually the bacilli are irregularly grouped together, but they may, especially the shorter forms, be arranged in pairs, and groups may be seen of several pairs disposed parallel to one another. They do not form spores. They can be artificially cultivated in various media. Their size and appearance depend considerably upon the medium and method of cultivation. There is another bacillus met with in cases of diphtheria, as well as in other throat affections. It is a short, wedge-shaped,



non-motile bacillus. The bacilli are arranged in groups of parallel pairs, the pairs having their bases in apposition. When stained it does not present a beaded appearance. This microbe does not vary with its culture medium to such a degree as the diphtheria bacillus. It is not pathogenic to animals. When grown in neutral broth it renders the medium alkaline from the first, whereas the diphtheria bacillus renders it firstly acid and then alkaline. This bacillus has been called Hoffmann's, the short diphtheria, and the pseudo-diphtheria bacillus.

**Bacteriological Investigation.**—A bacteriological examination for diagnostic purposes is made as follows.—With a sterilised cotton-wool swab, a loop of platinum wire, or, better still, the flattened end of a platinum rod, a small portion is removed from the edge of the exudation, or if none be present a scraping is made of the mucous surface. The swab or rod is then smeared slightly over the surface of sterilised blood-serum in a test-tube. The tube is placed in an incubator at 37° C. Colonies of diphtheria or of Hoffmann's bacilli can be seen in twelve to twenty-four hours as an opaque white streak or discrete points. Growth due to other organisms appear later, and are usually either transparent or not white. Occasionally, however, colonies resembling those of the diphtheria bacillus are produced by other organisms, so that no reliance as to the nature of the growth can be placed upon a naked-eye examination. As soon as it is convenient after the growth has become visible it should be microscopically examined. The bacilli may be stained by Gram's method, or with a solution of cubolic methylene blue, or other aniline dyes.

**Inoculation Effects.**—The cause causing diphtheria is the Klebs-Loeffler bacillus. It can be cultivated from the exudation in the vast majority of cases that are clinically diphtheria. When inoculated upon the mucous membranes of certain animals (e.g. guinea-pigs) it sets up an inflammation which may be membranous. When inoculated subcutaneously a local exudation results, around which there is extensive oedema. Necrosis of the tissues follows, and the animal wastes and dies in a few days. After death pleurisy, broncho-pneumonia, hemorrhage into the adrenal bodies, and degeneration of nerves and muscles are found. In cases where death is delayed beyond four or five days the animal has paralysis of the extremities. If the diphtheria bacillus is grown in broth under certain conditions, it produces in the broth bodies which by infiltration can be freed from bacilli. These bodies, according to Sidney Martin, consist chiefly of albumoses, with an organic acid. **Toxin.**—Collectively these bodies may be included under the term diphtheria-toxin. When the toxin is injected into a rabbit or guinea-pig it gives rise to, amongst other symptoms, paralysis of the extremities and trunk. Microscopically there

are degenerative changes in the nerves and muscles similar to those found in human beings who have died of diphtherial paralysis. From the blood and tissues (especially the spleen) of patients who have died of diphtheria similar toxic bodies can be extracted. It has already been stated that the diphtheria bacillus is as a rule confined to the local exudation. Hence the inference from the experimental evidence is that the bacillus manufactures toxic products locally, and these being absorbed into the system give rise to the degenerative changes so commonly met with. Martin, however, believes that the bacilli produce locally a ferment, which, being absorbed, acts on the proteins of the tissues and gives rise to the poisonous albumoses.

**Problems in the Pathology of the Disease.**—There are three points concerning the pathology of diphtheria which are worthy of notice. (1) With respect to the acceptance of the *Klebs-Loeffler bacillus* as the essential cause of diphtheria, it proves a stumbling-block to some persons that it is occasionally found not only in morbid conditions of the fauces and mouth other than diphtheria, but also in healthy throats. Therefore, they say, this bacillus cannot be the cause of diphtheria. To agree with these objectors, however, one would have to ignore absolutely the whole of the experimental evidence in its favour, which is not only exceedingly strong in itself, but has directly led up to the most successful method of treating the disease hitherto discovered. In order to its development the bacillus doubtless requires the influence of one or more favourable conditions, without which it possibly will not manifest any pathogenic action whatsoever. But, on the other hand, let all these conditions be present without the bacillus, and there will be no diphtheria.

(2) Another question of interest relates to the variation in virulence of the bacillus. The study of epidemic diphtheria points to the conclusion that the variation may be extreme, and that in the course of an epidemic it may be exalted from a low to a high degree of malignancy and *vice versa*. Roux and Yersin succeeded in attenuating the virulence of the bacillus by cultivating it in broth for a month at a temperature of 39.5° C. They were unable to restore the virulence when it had been completely destroyed, but when the virulence was only partially lost it could be regained by injecting into animals the *Streptococcus erysipellatus* along with the attenuated bacilli. It has been suggested by more than one bacteriologist that Hoffmann's bacillus is an attenuated form of the Klebs-Loeffler organism. But no convincing evidence of this suggestion has hitherto been brought forward. In order to prove that the former has really been converted into the latter organism it is required that from the broth in which subcultures of the changed bacillus have been grown the characteristic toxin of diphtheria

shall be extracted, a toxin not only capable of bringing about the nerve and muscle degeneration already described, but also neutralisable by the diphtheria antitoxin. And this has not yet been accomplished.

(m) Lastly, it must be mentioned that some authorities, especially the French, believe that the strepto- and staphylococci and other organisms so commonly found in the local exudation in a case of diphtheria have no small share in the pathology of the disease. They state not only that to these organisms are due such complications as adenitis, cellulitis, supuration, and so forth, but also that the virulence of the diphtheria bacilli is heightened by their presence. Hence they speak of "pure" and "associated" diphtheria, meaning by "pure" cases those in which a pure cultivation of the specific organism is obtained from the exudation, and by "associated" cases those in which the diphtheria bacilli are associated in the cultivation with a large number of other bacteria. While the writer shares this view in so far as to agree that in some cases certain of the complications mentioned can be attributed to the associated organisms, he cannot subscribe to the belief that they are all so caused, nor is he of the opinion that the "associated" is necessarily graver than the "pure" form. In his experience the clinical results of the antitoxin treatment are quite against this view. By merely examining a growth on serum or a slide under the microscope no knowledge is to be gained as to the severity of any given case.

**DIAGNOSIS.**—Since in faucial diphtheria in its earliest stage, and in many cases at a later period also, the exudation is slight and not membranous, and constitutional symptoms may be absent, it is easy to understand how difficult it is to distinguish the disease from other forms of sore throat, more especially *simple* and *follicular tonsillitis*. Every case of this kind should indeed be regarded with suspicion, and forthwith isolated and injected with antitoxin. A bacteriological examination should at the same time be made, and in the absence of any definite clinical symptoms the diagnosis should rest upon the result of this examination. It should, however, be borne in mind that one negative result is not sufficient upon which to pronounce an opinion that the case is not one of diphtheria. Of the two forms of tonsillitis above mentioned diphtheria assumes the simple more often than the follicular. Besides these the affections most commonly taken for diphtheria are *simple ulceration of the tonsils*, *catarrhal inflammation of the fauces*, and *scarlet fever with angina*. The very existence of ulceration (unless very superficial) is against diphtheria. In catarrhal sore throat the tonsils and the whole of the mucous membrane of the fauces are slightly swollen, and at first dry. Later there is an excess of mucous secretion.

Often the inflammation extends to the nasal passages (producing a discharge at first thin and watery, afterwards muco-purulent), larynx, and trachea (giving rise to hoarse cough and pain in the chest, rarely to urgent dyspnoea). There may be slight pyrexia, and the patient feels chilly and out of sorts. The condition is not infrequently met with in *influenza*.

*Scarlet fever* is often diagnosed as diphtheria, much less often is the latter affection mistaken for the former. The error arises, firstly, through the observer being unavoidably compelled to make a diagnosis before the appearance of the rash, secondly, through the omission to look for a rash, and, thirdly, through the absence of a rash. The onset of scarlet fever is usually accompanied by vomiting, which is not the rule in diphtheria. A high temperature, delirium, and marked swelling, with vivid redness of the fauces generally, are in favour of scarlet fever. But it is often impossible to distinguish a mild case of scarlet fever before the appearance of the rash from one of diphtheria. The rash may be absent or insignificant and transient not only in very slight cases of scarlet fever, but in a few severe cases with serious angina. These cases are a source of much trouble in diagnosis, for not infrequently there is thick pulsatious or even membranous exudation upon the fauces. In such cases the diagnosis cannot be made with certainty till a late period, when the exudation has cleared off, leaving behind deep or extensive ulceration, or until the characteristic desquamation has commenced. Anything beyond superficial ulceration is uncommon in diphtheria. In the cases of scarlet fever to which we are referring, suspicion is often roused before ulceration or gangrene has occurred by the irregular but continuous pyrexia (often with delirium), the persistence of the exudation (the more remarkable if antitoxin has been administered under the supposition of diphtheria), and the rapid emaciation of the patient. The larynx is rarely affected in scarlet fever, when this event does happen it is either secondary to intense cervical cellulitis, or it occurs later in cases where there is extensive and spreading faucial ulceration.

The writer has also known diphtheria to be mistaken for the following conditions:—*Erysipelas faucium*, quinsy, enteric fever, mumps, ulcerative stomatitis, thrush, syphilis (usually tertiary ulceration), tuberculous ulceration of the fauces, herpes of the palate, phlegmonia of the fauces (a rare disease), and not seldom, especially in infants, dried mucus and clotted milk adhering to the fauces. With respect to *erysipelas faucium* and *quinsy* the mistake is usually caused by the presence of exudation having the appearance of membrane. But in each of these diseases the initial shivering, high temperature, delirium, full and bounding pulse and intense swelling, brawny in the case of

erysipelas, followed by suppuration in quinsy, constitute a group of symptoms very different from those of diphtheria. In *enteric fever* the error is usually made in cases ushered in with tonsillitis, but it may be due to mistaking mspissated mucus, clotted milk, etc., for membrane. In some rare cases of *mumps* the cervical glands only are inflamed, and not the salivary glands, but in this disease the fauces are unaffected. Careful attention to the exact nature of the local lesions will prevent mistakes in the other diseases mentioned above. It has been incidentally stated that false membrane may be present in other diseases besides diphtheria (e.g. scarlet fever). It is known also that local irritation of a mucous surface by certain chemicals and by steam may excite a membranous inflammation. Other micro-organisms than the diphtheria bacillus may produce a similar result. But without careful bacteriological examinations it is impossible to distinguish these conditions, and, apart from such examinations, it is prudent to consider all membranous inflammation of the fauces to be diphtherial. The same remark is true of *membranous laryngitis*. In most cases of laryngeal diphtheria there is no difficulty with regard to diagnosis, since it follows or accompanies the faucial form of the disease. But when the affection commences in the larynx, or the faucial lesion is slight and transient, it is not easy to distinguish diphtherial croup from croup due to other causes. In some patients it is possible to obtain a view of the larynx with the laryngoscope. But this means is not always available, and it is useful to know that in most children the epiglottis can be brought into direct view for a moment by depressing the posterior part of the tongue, as the epiglottis is often affected in laryngeal diphtheria, membrane may in some instances be thus observed upon its tip or anterior surface. In other cases membrane may be coughed up. In all doubtful cases a bacteriological examination should be made, a scraping of the mucous membrane being taken as near the larynx as possible. Any affections causing laryngeal obstruction may be mistaken for diphtheria. But simple laryngitis, membranous laryngitis, and swellings external to the larynx give rise to most trouble in this respect. Of the latter, those which by bulging over the laryngeal orifice impede respiration are the most common, namely, in children (in whom the question of diagnosis from diphtheria chiefly arises), post-pharyngeal abscess, growths, and excessive hypertrophy of the tonsils. A careful local examination will usually clear up the case. Less commonly cases of a foreign body in the larynx, laryngismus stridulus, and ulceration of the larynx (especially the simple and the tuberculous) are wrongly diagnosed as diphtheria, while still more rarely croup due to paralysis of the abductors of the vocal cords, intra-laryngeal

growths, or oedema glottidis are thus mistaken. The history of a doubtful case should be carefully gone into, it will be observed that many of the diseases mentioned above run a course which is chronic compared with the duration of a case of diphtheria. It should be remembered that laryngitis may be the first symptom of an attack of *measles*. There is then usually a high temperature and frequent coughing and sneezing, with sometimes conjunctivitis and coryza.

The diagnosis of nasal diphtheria must often depend upon a bacteriological examination, since in many cases there is an absence of membrane. Vulval diphtheria may be simulated by erysipelas and noma vulvæ.

Diphtherial paralysis in cases where the primary sore throat has been slight or overlooked may be mistaken for some other form of peripheral nerve degeneration or neuritis, locomotor ataxia, or cerebellar tumour. But in the paralysis following diphtheria the soft palate is nearly always, and many muscles are frequently affected, there is rarely vomiting (apart from cardiac complications), and there is never optic neuritis or atrophy. On the other hand there is often some disturbance of the cardiac and respiratory rhythm.

**PROGNOSIS.**—The most important factors in prognosis are the age of the patient, the locality of the disease, and very often the duration of the disease before medical advice has been sought.

With respect to age the younger the patient the greater the probability of a fatal termination. As to locality those cases are most fatal in which the larynx is involved. Cases in which the nasal passages only are affected are the least fatal. In laryngeal diphtheria the gravest apprehension is to be entertained in very young children on the one hand and adults on the other. Vulval diphtheria is usually accompanied by severe constitutional symptoms. In ocular diphtheria the chief risk is to the eyeball.

The previous duration of the disease before medical advice has been taken has greater effect upon the prognosis in cases treated with antitoxin than in those not. The earlier the patient is injected with serum the more confidently can not only recovery, but recovery without the occurrence of serious complications, especially paralysis, be anticipated. Of particular symptoms the following are very ominous.—Frequent vomiting or retching, a pulse-rate falling to below 50 per minute, marked cardiac irregularity, hæmorrhages from mucous membranes and, even though few, into the skin and subcutaneous tissue, suppression of urine, convulsions.

Those cases in which there is extensive and persistent local exudation, with much albuminuria, furnish the most severe cases of paralysis. The more widely spread the paralysis, and the more rapidly it becomes generalised, the greater is the danger to life. Affection of the respiratory muscles is especially dangerous. Attacks of

syncope, frequent vomiting, and severe epigastric pain occurring during the course of paralysis are all grave symptoms. In non-fatal cases of paralysis recovery is always complete.

#### TREATMENT

By far the most satisfactory method is that known as the *antitoxin treatment*, which we owe chiefly to Behring and Kitasato. Hence we shall deal with it first. But before entering into details a few facts indicative of its value will not be out of place.

The treatment was tried, in the first instance, in Germany in 1893, but not until after Roux's paper at the Hygiene Congress held at Budapest in September 1894 was it extensively practised in other countries. Since that date there has come from all parts of the world an almost unanimous agreement of opinion in its favour. The results of the first series of cases treated in the United Kingdom were brought before the Clinical Society of London in December 1894 by Dr. Washbourn, Mr. Curd, and the writer. They showed that the mortality of the cases in the Eastern Hospital, Hoxton, of the Metropolitan Asylums Board, was at once reduced to half what it had previously been, and reported most favourably upon the treatment, an opinion which was fully endorsed by a Special Committee of the Clinical Society. The treatment was subsequently introduced into other hospitals, notably those of the Asylums Board. The following figures show with what results—

CASE-MORTALITY OF DIPHTHERIA IN THE HOSPITALS OF THE METROPOLITAN ASYLUMS BOARD

Year	1892	1893	1894	1895	1896	1897	1898
Mortality per cent	29.5	30.4	29.2	22.5	21.2	17.6	15.5
Percentage of cases treated with antitoxin	—	—	—	61.5	66.2	80.2	81.4

That these results are not accidental may be shown in more than one way. For instance, in London, up to the end of 1897 at any rate, antitoxin was used but little outside the Asylums Board Hospitals. Consequently there was very little lowering of the mortality of cases not admitted to those Hospitals. Again, there has been a very marked improvement in the laryngeal cases, among which the number included merely upon bacteriological evidence is very small. The writer has elsewhere shown that whereas in the pre-antitoxin days there was small expectation of saving more than 29 per cent of the tracheotomies, with antitoxin recovery is to be looked for in no fewer than 53 per cent. A similar improvement has taken place with respect to the cases not operated upon. Clinically the beneficial results of antitoxin appear as follows: the exudation does not spread or re-form, that

which is already present clears off more quickly than in the cases not submitted to antitoxin, and the foul nasal discharge rapidly dries up. Consequently the patient is enabled to breathe and swallow with comfort, and his general condition improves. The cervical adenitis and cellulitis subside, and the temperature and pulse-rate fall. It is rare for the larynx to become invaded after antitoxin has been injected, so that extension of membrane to the lungs is very much less common than it used to be. Fatal broncho-pneumonia is also less frequent. Lastly, in cases treated early, on the first or second day, paralysis is much less likely to follow than in cases treated later or without antitoxin.

*Basis of the Antitoxic Treatment*—The antitoxic treatment is based upon the following facts. It has been experimentally proved in a general way that the blood-serum of an animal immunised against a certain micro-organism and its toxin has the remarkable property of acting as an antidote to both the micro-organism and the toxin. There are several methods of producing artificial immunity. One of them consists in injecting into an animal at intervals of a few days gradually increasing doses of the toxin produced by the growth in broth of the organism immunity to which is sought. After a time it will be found not only that the animal is able to bear without ill effects the injection both of the living organism itself and of its toxin in large and otherwise fatal doses, but also that its blood-serum has antidotal properties. If a sufficient quantity of the serum be mixed with a fatal dose of the toxin and the mixture be injected into a non-immunised susceptible animal, the latter will not suffer from any of the ill effects that would follow the injection of the toxin alone. The special application of these general principles has been rewarded with most success in the case of diphtheria. In order to produce the antidotal or antitoxic serum the horse is the animal selected, partly because its size allows a large yield of serum, and partly because it bears well the process of immunisation. The serum having been obtained, the next step is to estimate its antitoxic value.

Formerly this was ascertained by testing the serum with broth containing toxin, it being assumed that amounts of different broths that were equal in their toxicity were also equal in their power of combining with antitoxin to form a harmless compound. But Ehrlich has shown that this is not the case. He states that all freshly-prepared broths contain not only toxin, but also a body termed by him *toxone*, which, while it has the combining, is largely wanting in the toxic power of toxin. Further, when the broth is kept for some time, part of the toxin becomes changed into bodies which have properties similar to those possessed by toxins, and which are termed *toxoids*. Hence it follows

that not only do different broths vary from one another, but also that the same broth varies from time to time with respect to the relative proportion of its toxic and non-toxic combining powers. The instability of toxin has prevented the production of a standard toxin for general use. Antitoxin, however, is a much more stable body. By a series of experiments (which he states can be accurately repeated) Ehrlich believes he has succeeded in eliminating the errors due to the varying constitution of toxin-broth, and has thus been able to obtain an antitoxin of uniform strength. This antitoxin is now used in most laboratories for testing a new serum. It is, firstly, ascertained what quantity of a toxin produced in the usual way is neutralised by one unit of Ehrlich's standard antitoxin. Secondly, it is determined how much of the serum, of which the antitoxic value is being tested, will neutralise this ascertained quantity of toxin. The definition of a "unit" is as follows:—"A serum of which 1 c c when mixed with 100 times the fatal dose of toxin protects a guinea-pig of about 250 grammes weight from death within ten days, contains one unit per c c." Park points out that in this definition toxin must be taken to mean a toxin "having the characteristics of toxins in culture at the height of their toxicity."

At the present time sera can be obtained which contain as many as 4000 units in 5 to 10 c c.

Before we proceed to discuss the question of the usage of the serum in the human subject we must draw attention to one very important point in the experimental evidence. It has been found that if an interval be allowed to elapse between the injection of the toxin, and subsequently the antitoxin, into a susceptible animal, then the longer the interval the less effectual will be the action of the antitoxin, and, finally, there comes an occasion when the interval has been made too long, and the antitoxin is injected too late to prevent the lethal effects of the toxin. Hence it was predicted by Behring that the success of the treatment would be found to depend very largely upon the earliness of its application. Clinical evidence has amply borne out this prediction. The following figures, illustrative of this point, are taken from the Statistical Reports of the Metropolitan Asylums Board:—

Day of disease upon which patient was admitted (1894) or brought under antitoxin treatment (1896-97)		1st	2nd	3rd	4th	5th and later
Non antitoxin cases 1894, all the hospitals	Cases	189	599	652	566	1152
	Deaths	90	146	192	179	355
Mortality		22.5	27.0	29.4	31.6	30.8
Antitoxin cases, all the hospitals (1895-96) with Brook and Eastern Hospitals only for 1897	Cases	209	1126	1113	1332	2436
	Deaths	8	147	275	376	780
	Mortality	3.8	12.1	20.9	29.2	32.0

Further, the mortality of the cases of diphtheria occurring among the patients convalescing from scarlet fever in the Asylums Board Hospitals has been reduced from an average of 50 per cent to 5 per cent. Nearly the whole of these cases are brought under treatment on the first or second day of their illness.

Unfortunately a very large proportion of the patients admitted to the Asylums Board Hospitals have been ill longer than three days. What can be done by the early administration of antitoxin amongst even the poorest patients treated in their own homes has been shown by the Department of Health for the City of Chicago. In that city prior to the antitoxic period the average case-mortality in hospital and private practice was about 35 per cent. In October 1895 the Department undertook to supply antitoxin gratuitously to the poor, a medical officer being sent to administer it immediately upon receipt of the notification of the case. This action was at once followed by a most extraordinary and gratifying fall in the case-mortality, which was for the period 5th October 1895 to 31st December 1896, 6.57 per cent, for 1897, 6.93, for 1898, 7.33, and for January and February 1899, 6.53.

**DETAILS OF ANTITOXIN TREATMENT**—Inasmuch as in diphtheria the disease has already got a start of the remedy, the mortality will probably never be reduced to zero. But the above and other similar figures go to show that if antitoxic serum were employed not later than the second day of the disease the case-mortality would not exceed 10 per cent. Therefore the cardinal rule in the antitoxic treatment is—Inject early.

If the treatment is commenced on the first day the dose should be 1500 units at least, it will usually be unnecessary to give more than 2000. But if it be delayed, the amount must be increased up to 8000 or 10,000 units, according to the severity of the case. It is advisable to repeat from half to the whole first dose within twenty-four hours if the local exudation shows no sign of resolution. With respect to the total amount to be administered, though as far as the writer knows (and he has often injected from 30,000 to 50,000 units) the limit is set only by the volume of the serum that can with convenience be injected, yet his experience leads him to say that little is to be gained by giving more than 16,000 units during the first twenty-four hours from the commencement of the treatment. But, again, the earlier the treatment is begun, the less necessity will there be for large and repeated doses. As the more concentrated sera are more expensive than the less concentrated, early treatment is more economical.

The serum is administered by injection into the subcutaneous tissue of the flank or abdomen. The skin at the seat of injection should be

previously washed and cleansed with some antiseptic solution. The most convenient form of syringe is Roux's. It can be readily taken to pieces and boiled to render it aseptic, it does not easily get out of order, and it has an ingenious device for tightening up or loosening the india-rubber plunger. In this country it is unnecessary to have the glass barrel or the piston-rod graduated, since the serum is given by the unit, and not by the cubic centimetre or minimum. The needle is connected to the nozzle of the syringe by a piece of rubber tubing. Care should be taken not to inject air with the serum. All the parts of the syringe should be boiled immediately before use. After the injection the small puncture made in the skin should be sealed with collodion. For cleansing the syringe after use cold water should be employed, for if the syringe be plunged into hot water the needle, tube, and nozzle will become blocked with coagulated serum, the removal of which will give much trouble.

*After-effects*.—There are certain effects due to the serum with which it is necessary to be acquainted. The most common are erythematous rashes, inflammation of or about joints, and abscess at the seat of injection. Rashes occur in from 30 to 45 per cent of the cases. The most frequent are erythema multiforme and urticaria. The rash usually appears seven to twelve days after the injection, but it may come out as early as the first day or as late as the third week. In many cases there are also febrile symptoms. The rash often commences at the seat of injection, its duration varies from a few hours to several days. Arthritis and peri-arthritis occur in about 6 per cent of the cases. They come on about the same time as the rash. The knees, hips, shoulders, elbow, and wrists are most often affected, but any of the joints may be involved. The arthritis lasts from one to four days. It is usually accompanied by one of the rashes above mentioned and pyrexia. Peri- or endocarditis does not occur. In most cases there is no swelling of the joints, and pain is the only symptom. Very often the pain is more in the large fasciæ and tendons than the joints. An abscess at the site of injection usually means that the technique of administration has been faultily performed, provided that the serum be sterile, as it should be. But possibly in some instances of mixed infection (e.g. diphtheria and scarlet fever) the presence of septic micro-organisms in the blood determines the local suppuration. In very rare cases other sequelæ have been met with, œdema of the serotum with or without slight oedema, and rigors, pyrexia, rapid pulse, and prostration coming on immediately after an injection, sometimes with a rash. The cases of rigors, etc., observed by the writer have all occurred in patients treated a second time with antitoxin in

a relapse or second attack of diphtheria. The rashes and joint-pains, but not the abscesses (which are caused by cocci), are due to the serum, and not to the antitoxic principle in it, for they can be produced by the injection of the serum of a normal non-immunised horse. Their occurrence is determined partly by the idiosyncrasy of the patient, but mostly by that of the horse from which the serum has been obtained, for the frequency varies with different sera. Generally speaking, however, the larger the volume of serum injected the more likely is a rash or arthritis to occur. Therefore with concentrated sera these troubles are usually at their minimum. In most of the cases they are trivial. It is possible that the serum also produces transient albuminuria, but it does not cause nephritis or suppression of urine. The writer has indeed on several occasions injected serum into patients, the subjects of acute or chronic nephritis, without any ill effects.

Serum will remain efficient for several months, but it is advisable not to keep it for more than a few weeks, as there is reason to believe that in some instances the antitoxic value becomes lowered. It should be kept in a cool and dark place. Never keep a bottle of serum which has been uncoiled.

*Local Treatment*.—In addition to antitoxin local treatment is advisable in most cases, in order to remove the exudation and render clear the obstructed faucial and nasal passages. For this purpose the affected regions should be frequently flushed out with warm water, or a saturated solution of boracic acid, or the following solution, *Sodii bicarb* ʒj, *sodii bihor* ʒj, *sodii chlorid* ʒss, *potassii chlorat* ʒss, *tinct. lavand. comp* ʒj, *aq. ad* Oj. The flushing out is best accomplished by means of a Higginson's or a ball syringe. But it, as is often the case, the child violently resists all attempts at local irrigation, it is advisable not to persist, for such attempts only exhaust the patient and do more harm than good. The use of solutions containing such agents as chlorine, sulphurous or carbolic acid, which act as irritants to the mucous membranes, are, in the writer's opinion, undesirable, except in very foul and septic cases.

The best local treatment for ocular, vulval, and cutaneous diphtheria is frequent irrigations and warm fomentations of boracic acid solution.

When laryngeal symptoms arise, the patient should be placed in a room, or, if in a ward, a tent, in which the air is kept saturated with the warm vapour of water by a steam-kettle. In many cases the symptoms will, under the antitoxin treatment, subside without any question of operative interference arising. But should there be cyanosis, restlessness, much recession of the chest-walls (which leads to pulmonary collapse), or frequently recurring acute attacks of dyspnoea, relief must be afforded by intubation or tracheotomy.

<sup>1</sup> See "Drug Eruptions."

**Intubation and Tracheotomy**—In this country intubation in diphtheria has never attained to the favour that has been accorded it in the United States and on the Continent, where since the introduction of the antitoxin treatment it has almost superseded tracheotomy. This difference in practice is very largely due, in the writer's opinion, partly to the fact that abroad many cases are intubated which here are not operated upon in any way, and indeed recover without any need for operation, and partly to the fact that the patients are brought under the antitoxin treatment at an earlier stage of the disease than in this country. The advantages urged on behalf of intubation are that a cutting operation is avoided, together with all the risks of tracheotomy (emphysema, hemorrhage, broncho-pneumonia, etc.), that no anæsthetic is required, that especially if repeated it acts mechanically in clearing out the larynx by the removal of membrane, and that its results are more successful than those of tracheotomy. The probable cause of the last statement is alluded to above. On the other hand, an intubated child must never be left out of immediate reach of a medical man, the tube may be suddenly coughed out, and the urgent symptoms of obstruction recur, or the tube may become blocked while in the larynx. The nurse is usually quite helpless should either of these events happen. The tube sometimes sets up ulceration of the larynx. Bumbling and forcible attempts to introduce the tube will damage the larynx, and cause false passages. The writer has seen all these accidents except the last. With respect to difficulty in execution there is little, if any, difference between intubation and tracheotomy. The writer's experience leads him to formulate the following propositions—In a considerable proportion of cases intubation is either inadmissible or inexpedient. It is admissible in two classes of case—those in which the patients, when brought for treatment, are *in extremis* from suffocation, and those where there is very abundant faucal membrane. It is inexpedient in toxic cases in which there is little or no hope of the patient's recovery. Here tracheotomy should be performed to ease the patient's last moments. Otherwise intubation should be practised. The intubation tube should be taken out at the end of two days. If it has then to be replaced it should be taken out at the end of another two days, and if necessary again replaced. If, at the end of two more days, withdrawal of the tube still leaves the patient in distress, it is best to tracheotomise. In cases where the tube is repeatedly coughed out it is not advisable to intubate more than six or seven times. Lastly, if there is evidence of extension of membrane below the larynx, tracheotomy is to be preferred to intubation. It is stated by some writers that membrane is coughed up through the intubation

tube, but the writer has never met with such a case. None of the cases he has had under observation, in which there was membrane below the larynx, have done well with intubation, but have come sooner or later to tracheotomy. In private practice tracheotomy is certainly to be preferred to intubation, and the operation should be resorted to earlier than in hospital practice, where both operator and instruments are at hand for any emergency. For the details of the two operations, etc., the reader is referred to the articles dealing with them (*See "Intubation," "Tracheotomy"*).

**General, Dietetic, and Medicinal Treatment**—During the acute stage of diphtheria the patient should be kept at rest in bed, and he should not be allowed to get up for some days after the disappearance of the local exudation, and only then if the circulation is normal and there is no sign of paralysis. Cases that present symptoms of toxæmia (albuminuria, vomiting, frequent and irregular pulse, etc.) require most careful watching. Serious and even fatal attacks of syncope may be induced by allowing a patient to get up and walk about too soon. Caution has to be exercised in letting the patient even sit up in bed.

The diet calls for no special remark. It should be such as the patient can take with comfort, frequently administered in small quantities at a time. Repeated vomiting at any stage of the disease is best met by rectal feeding. Alcohol and strychnine are useful in the late but not the early cardiac failure, more especially in attacks of syncope. The anemia, too, often following the disease usually calls for iron in some form. Fresh air and sunshine are very necessary for rapid convalescence.

At the first sign of paralysis (nasal voice, regurgitation of food through the nose, etc.) the patient, if up, should be again confined to bed. If after a week or ten days the palsy remains limited, he may be allowed up again, but should be warned against or prevented from exerting himself. Any advance of the paralysis is an indication for further rest. In fact, complete rest is essential in all cases where the palsy is at all widely spread. In such cases all excitement (*e.g.* visits from relations and friends) should as far as possible be avoided. When the respiratory muscles are affected the foot of the bed should be raised to allow the bronchial secretion to escape into the mouth, and to prevent the saliva trickling through the larynx into the bronchi, otherwise the lungs become blocked to such a degree as to lead to a fatal result. Extract of belladonna,  $\frac{1}{2}$  grain every three or four hours, is recommended in order to diminish the amount of these secretions. A cough during swallowing is always an indication to administer nourishment by means of a tube passed through the nose into the stomach.

**Isolation and Disinfection**—The patient suffer-

ing from diphtheria should be isolated. If he is kept at home all precautions should be taken to prevent the transmission of infection to others. As a rule, he should be kept in isolation for at least four weeks from the commencement of the illness. In the case of children the writer prefers isolation of at least six weeks. Even then they should not be allowed to mingle with healthy children at the end of that time if there is any discharge from the nose or inflammatory condition of the fauces. The so-called "return" cases are not so common in connection with this disease as with scarlet fever. It has been stated by Park and others that as a rule the diphtheria bacilli are absent from the fauces three weeks after the disappearance of the exudation. But in the writer's experience cases have not infrequently occurred in which the organism has been present in a virulent form for several weeks or months after an attack of diphtheria, and that in spite of local antiseptic treatment. The question arises whether such a patient is to be kept isolated as long as the bacilli are present. On this point the writer can only say that he has not detained in hospital such cases for a longer period than three months, and some even for a shorter time, and to the best of his knowledge no secondary or "return" cases have been caused by such patients after their discharge. At the same time in letting such patients out of isolation it is advisable to impress upon the parents or friends the wisdom of keeping the recovered patient as far as possible to himself with respect to other children (e.g. not sleep with children, kiss them, etc.), and to be on the look-out for any recrudescence of sore throat.

**Prophylaxis.**—Another question which arises in the prophylaxis of diphtheria is: Should antitoxin be employed as a preventive agent? As a rule the writer would answer, No. The prophylactic power of antitoxin does not last for more than three weeks, if so long. But there are a few well-reported and authentic instances where a case of diphtheria having occurred in an institution for children (hospital, school, etc.), and other cases having followed at intervals, the outbreak has been cut short apparently by submitting the remaining children to the injection of antitoxin. Therefore in special instances this measure is worth a trial. It is not necessary when injecting antitoxin as a prophylactic to give more than 600 units.

**Diphthongia or Dipthongia.**—The production of two notes of different pitch, a form of vocal disturbance met with in some morbid states of the vocal cords. See LARYNX, BENIGN GROWTHS OF (*Clinical Features*).

**Diapacusis.**—An incorrect hearing, two tones being heard when one only is produced. See AUDITORY NERVE AND LABYRINTH (*General Diagnosis*), EAR, MIDDLE, CHRONIC NON-SUP-

PURATIVE DISEASE (*Hyperthrophic Catarrh, Dys-harmonic Diplococcus*).

**Diplegia.**—Paralysis of corresponding parts on both sides of the body, e.g. of both legs, of both arms, or of both sides of the face. See PARALYSIS (*Cerebral Diplegia*). See also MENTAL DEFICIENCY (*Accidental, Acquired, Traumatic*).

**Dipleural.**—Bilateral (Gr. *dis*, double, *πλευρόν*, the side).

**Diplocephalus.** See DICERPHALUS.

**Diplococcus.**—A double micrococcus or a pair of micrococci joined together as a dumb-bell-shaped organism, such as the *D. intracellularis meningitidis*, the *D. albicans amplus*, etc. See ACNE (*Acne Vulgaris, Micro-Organisms*), MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Bacteriology*), NOSE, ACCESSORY SINUSES, INFLAMMATION (*Bacteriology, Diplococcus Pneumoniae*), SUPPURATION (*Etiology, Diplococcus Pneumoniae*), TUBERCULOSIS (*Mixed Infection in*).

**Diplocoria.**—Double pupil, e.g. that due to subdivision of the pupil by a band of persistent pupillary membrane, polyconia.

**Diploe.**—The cancellated substance found between the inner and outer tables of compact tissue of the bones of the cranium, it contains thin-walled, branching, valveless veins, and it is absent in the orbital plates of the frontal and the cribriform plate of the ethmoid bone.

**Diplogenesi.**—The formation of a double monster or poly-somatous teraton, diplo-teratology.

**Diploma.**—A document granted by a university or college, "testifying to a degree taken by a person, and conferring upon him the rights and privileges of such a degree," e.g. to practise medicine. "The granting of diplomas by universities or other learned bodies proceeds on the supposition that the public require some assistance to their judgment in the choice of professional services, and that such an official scrutiny into the qualifications of practitioners is a useful security against the imposture or incompetency of mere pretenders to skill" (Illustr. quot. in Murray's *New English Dictionary*).

**Diplomyelia.**—A double condition, due to antenatal causes, of the spinal cord, diastematomyelia.

**Diplopi.**—The seeing of single objects as double, double vision, it may be binocular, disappearing when one eye is shut, or (rarely) unocular or monocular, when it is binocular it may be homonymous (the position of the double images corresponding to the position of the eyes), or heteronymous or crossed (the left



image corresponding to the right eye, and the right image to the left eye), a common cause is squint. See HYSTERIA (*Ocular Symptoms*), LACRIMAL APPARATUS (*Diseases of Gland, Chronic Adenitis*), MYANTHENTIA GRAVIS (*Symptomatology*), NOSE, ACCESSORY SINUSES, INFLAMMATION (*Dipnomia, Eye-Symptoms*), OCULAR MUSCLES, AFFECTIONS (*Paralysis*), SPASMUS, SYPHILIS (*Tertiary, Eye-Symptoms*), THYROID GLAND, MEDICAL (*Exophthalmic Goitre, Symptoms*)

**Diplosomus.**—A double monster or united twins in which there are two bodies, the heads being more or less fused

**Diploteratology.**—The science of double monsters, their characters, classification, and mode of production

**Diprosopus.**—The teratological condition in which there are two faces, more or less fused into one, there may be four eyes, or three (one of which is a fused double eye), or two, and four ears or three

**Dipsesis.**—Excessive thirst (Gr *δίψαω*, I thirst)

### Dipsomania.

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See also ALCOHOLIC INSANITY (*Definition*), HYPOTISM (*Therapeutic Uses, Dipsomania*), INSANITY, NATURE AND SYMPTOMS (*Impulse and Obsession, Dipsomania*)

**DEFINITION.**—Dipsomania is an elaborate idiosyncrasy towards alcohol with occasional paroxysmal and irresistible impulses to drink excessively

**SYMPTOMS.**—Dipsomania is, *par excellence*, the alcoholic disease of good people. Both men and women are the victims of it, but women less frequently. The idiosyncrasy which it betokens is a congenital equilibrium, and occurs generally, but not always, in obviously neurotic subjects. The paroxysmal phase usually develops for the first time in the second half of adolescence, but may occur any time after the patient has tasted alcohol and has discovered his susceptibility to it. True dipsomania is comparatively rare.

One cannot insist too strongly upon the fact that dipsomania is not the development of an alcoholic habit. It is much more truly the exact opposite—an abrupt and occasional departure from habit. We have to suppose—though we cannot always obtain evidence of it—that the patient, on having tasted alcohol, has been aware of an excessive and peculiar reaction to it. Not that he has become rapidly intoxicated, or even that anything which an onlooker might observe has occurred, but that the patient has

discovered in alcohol a source of acute, expansive pleasure. If he be a very strong man, the paroxysmal impulse may be indefinitely postponed. In my opinion there are quite a number of patients in whom the disease never becomes actual. They carefully avoid what is to them a very dangerous indulgence. More frequently, however, the patient does not deny himself so strictly, and occasional debilitate indulgences strengthen, in his mind, the impression of delight in drinking. Some cases have been described in which, almost from the first, there was no effective check upon drinking, but the usual thing is for the patient to resist his vice successfully for a considerable period. Sooner or later, however, in typical cases, the paroxysmal phase occurs—a quite unique and unmistakable development. The usual clinical picture is as follows:—

On some occasion of fatigue—towards the end of a busy season, under the depression of bodily illness or mental stress, during lactation, or in the reaction which occurs in busy people who suddenly find themselves at leisure—something very like an attack of acute melancholia sets in. The patient goes off his sleep and loses appetite, becomes costive, and has a dry skin, and dry, furied tongue, suffers some cardiac discomfort or pain, and some mild degree of dyspnoea. The characteristic feeling which persists is one of acute apprehension. This preliminary stage is short—often not more than from six to forty-eight hours. Then occurs the alcoholic obsession. The patient becomes aware that the idea of drinking is occurring to his mind with unpleasant frequency. Do what he will to draw his attention to some projects, he finds the task impossible, and, a few hours later, the alcoholic idea is constant in his mind, and his interest in other things almost completely in abeyance. Still acutely apprehensive, the patient now finds himself struggling against a tyrannous impulse. The stage of obsession is past. With some ingritive thoughts of what he is drifting away from—home, business, honour—the patient resigns himself to the imminence of the alcoholic idea. Then he passes from the contemplative to the purposeful suggestion. His struggle now is not with his thoughts, but with his impulse. He is entirely and vainly occupied in trying to compel himself to sit still or to go to a safe place, in trying to resist the ungovernable impulse to go to the wine cellar or the public-house.

This may seem a common occurrence, a mere account of what temptation is to every man who tries to break a bad habit. But no one who has seen a dipsomaniac in the phase of impulse will easily forget the picture, or confuse it with the ordinary features of alcoholism. The general impression suggests the idea of an instinct. One is reminded of how animals behave under the intense excitement of the

reproductive or the maternal instinct, the instinctive fear for a natural enemy, the instinctive hunger for a natural prey. The whole man is changed. His face is hard and set, full of fear, evidently bent upon something—either a way of escape or a means of satisfying his craving,—his muscles are twitching, he is restless and under great bodily distress, his respiration is panting, his pulse quick, his skin hot, his tongue dry and parched. Actual thirst is acute, but, though the patient drinks large quantities of bland liquid, satisfaction does not follow, and cannot, except by intoxication. The characteristic termination of the attack is by some degree of coma. Thus the patient accomplishes rapidly once he resigns himself to it. And now it is characteristic that he drinks alcoholic liquor as a water-starved man drinks water—not with any lingering pleasure, but in drenching doses. At this stage, when the patient has begun to drink, volition practically ceases to exist except for purposes of drinking, and he is dangerous to himself and to others. Any one who tries to thwart him may be violently assailed, the most dangerous acts of escape from confinement may be attempted, and even extreme acts of immorality will not daunt a patient whose mind is made up.

By degrees—often not so quickly as would happen in a normal man—some degree of coma follows. Unless the liquor has been diluted there is risk of alcoholic poisoning. When the patient is so comatose as to be oblivious of all else, he will still reach out for drink and pour it down his throat. It is not usual for positive, alcoholic symptoms—excitement, delirium, convulsions, and such like—to be prominent in the first bout. The patient at last sleeps, and awakes, as a rule, feeling better. Very often, however, there is only an interval of peace, and relapses are very common. Probably after a week or ten days of repeated bouts the patient recovers by prostration, a condition of collapse in which he is free from the alcoholic suggestion, or merely delirium tremens or insanity. It is important to note that patients who have been prevented survive a paroxysm with a self-respect which has been retained at the cost of organic satisfaction, and are liable to have recurrent paroxysms. Those who drink, while they may be plunged, when they recover, into remorseful distress, have a sense of satiety which is, to some extent, a safeguard.

The subsequent history of cases is varied. Generally speaking, paroxysms are not periodic, but are more truthfully to be described as occasional. Some cases become rapidly worse, others seem to pass through one paroxysm after another without much depreciation. Circumstances which have been the occasion of a paroxysm are apt, if they recur, to occasion another and another—probably by suggestion. But many cases which have been treated judi-

ciously outgrow their idiosyncrasy, or at least escape its paroxysmal phases.

**NATURE AND PATHOLOGY.**—The pathology of dipsomania is quite unknown. To understand the condition it is helpful to consider the two phases of the disease—the idiosyncrasy which predisposes to it, and the paroxysm which expresses it. I would revert again to the analogy of a brute instinct. There is something quite unusual in the patient's reaction to alcohol, comparable perhaps to the constitutional effects of characteristic odours upon the lower animals. We perhaps go too far in describing the reaction as pleasant, but it must obviously be at least attractive. As a matter of fact, however, the patient is often not sure whether he more likes or dreads the experience which follows upon a mild indulgence. The condition of the patient in the subsequent stage—the stage of resistance—must also be interpreted in the light of his previous experience. Probably the patient's imagination is more habitually under the influence of the alcoholic idea than he confesses. The paroxysm may be variously explained. Perhaps it is a crisis—the culmination of a progressive, alcoholic psychosis comparable to an epilepsy. Or it may simply be that persons who have the alcoholic equilibrium which dipsomania betokens are liable to occasional recurrent attacks of acute melancholia, and that then the alcoholic suggestion which has been suppressed obtains a rapid and complete ascendancy. Whatever the morbid process by which we may suppose to underlie the phenomenon, dipsomania is anervous condition in which there is (1) a nervous state in which alcoholic stimulation is powerfully suggestive, (2) occasional conditions in which the alcoholic neurosis assumes an epileptoid intensity, or in which the volitional functions generally are so reduced as to admit of its rapid development. The forcefulness of the impulse is nothing new. We are familiar with the same kind of thing in other pathological states—pyromania, nymphomania, etc., and, as I have said, in brute instinct. But no one has a satisfactory explanation of such phenomena.

**DIAGNOSIS.**—It is the common custom to call all manner of cases by the name dipsomania which have not much in common with it except just the bouts of excessive drinking. Such a practice may not be of very evil consequence, but it is far from exact. True dipsomania cannot easily be mistaken. The condition is constitutional and native, it does not develop in consequence of alcoholic habits, and it has dramatic features which are impressive and unique. Some writers speak of pseudo-dipsomaniacs, but it seems to me better to consider these as varieties of alcoholism. Features which characterise dipsomania—the unusual reaction, the occasional crisis, the forceful impulse—may each of them occur in alcoholism. But unless

the patient manifests these symptoms independently of persistent alcoholic habits, he is not a dipsomaniac. Moreover, in dipsomania the symptoms common to alcoholism (*q v*) do not abound. Apart from these cases of alcoholism dipsomania is, as I have said, rare.

**ETIOLOGY.**—Dipsomania is not hereditary in the strict sense. It is an inequilibrium which may arise from any neuropathic family, and it may be the first instance, in a long race, of nervous degeneration. The occasions of paroxysmal impulse are of much more practical importance than are the constitutional causes of the idiosyncrasy. These have already been indicated. Adolescence is of the first importance, in older men, the preclimacteric stage when business is often excessive, and, in women, the late puerperal and lactational period. As in other neuroses, the periodicity of the paroxysms is more marked in women than in men.

**TREATMENT.**—The treatment of dipsomania is palliative as regards the idiosyncrasy, and preventive as regards the paroxysm. In other words, the patient should be treated as neurotic, and discipline and hygiene should be made to minister to stability. The treatment usually prescribed refers almost entirely to the prevention of drinking when a paroxysm has occurred. It is questionable if that is important. It is much more valuable to anticipate the crisis. When a patient is expecting an attack, active steps should be taken to outflank it. An abrupt and sudden readjustment of the whole circumstances of the patient is called for always in the direction of recuperation of energy. Change is the first essential, and it must be borne in mind that very often an unusual activity is recuperative. If the paroxysm has not been forestalled, and the physician is called to a case in which the stage of obsession or that of impulse has been reached, he must devote himself to the least hurtful satisfaction of the craving. An enema should be administered, the stomach should be washed out, and sedatives or motorics should be administered by the tube. At the same time nutrients should be added, and saline or other aperients. It must be the physician's aim to induce hypnosis as speedily as possible, and at the same time to further elimination. The patient must, in no case, be told what has been administered. Hypnotism is very useful if resorted to soon enough, but is not likely to be applicable in the paroxysmal stage.

**Dipsophobia.**—"Fanatic abstinence from intoxicating liquors." See **INSANITY, NATURE AND SYMPTOMS** (*Insane Defects of Inhibition*).

**Diptera.** See **MYIASIS** (*Myriasis Intestinalis, Diptera Larvæ*), **PARASITES** (*Insects, Diptera*).

**Dipus.**—Double foot, as in cases in which there are from seven to nine digits on one foot, along with indications that the foot is really two feet more or less completely fused; the term *dipus* is also given to double monsters in which there are only two feet (instead of three or four).

**Dipygus.**—That type of double monstrosity or united twins in which the lower end of the trunk is double (double sacrum).

**Dipylidium Caninum.** See **PARASITES** (*Cestodes, Tænia Canina*)—A common intestinal parasite in the dog, the ripe proglottides have the shape of melon seeds, hence the synonymous name *Tænia cucumerina* sometimes given to it, the larvae develop in the lice and fleas of the dog.

**Direct Action.**—The effect which a drug produces on an organ such as the kidney from contact, local action (see **PHARMACOLOGY**) in contrast to indirect action, which is a secondary effect.

**Direct Tracts.** See **SPINAL CORD** (*Anatomical Considerations*), **PHYSIOLOGY, NERVOUS SYSTEM** (*Spinal Cord, Conducting Paths*).

**Director.**—An instrument, grooved on one side, for directing the course of a knife or scissors in making an incision into an organ, an abscess, or a cyst in the midst of important structures which it is very desirable not to wound.

**Dirrhinus.**—A rare form of monstrosity in which there are two noses (Ballantyne's *Antenatal Pathology*, vol. II p. 397).

**Dirt-Eating.** See **PICA**.

**Disaccharids.**—Double sugars, *e g* maltose, two glucose molecules, polymerise to form one maltose molecule. See **PHYSIOLOGY, FOOD AND DIGESTION** (*Carbohydrates*).

**Disassimilation.**—The transformation of assimilated substances in the body into less complex compounds with liberation of energy, catabolism or katabolism, downward metabolism. See **PHYSIOLOGY, PROTOPLASM** (*Metabolism, Katabolic Changes*).

**Disassociation.**—The breaking up of a complex molecule into simpler ones at a certain elevation of temperature (*temperature of disassociation*), with reunion of the simpler molecules afterwards when the temperature falls.

**Disc.**—Any rounded, plate-like body, but especially the *optic disc* (entrance of optic nerve, or blind-spot), *blood discs* (red blood corpuscles), *Bowman's* and *Hensen's discs* (the discs into

which a muscular fibre can be split up), *inter-vertebral discs* (intervertebral cartilages in the spine), *tactile discs*, etc. See RETINA AND OPTIC NERVE (*Inflammation of Optic Nerve*, "*Choked Disc*"), etc

**Discharge.**—A secretion or excretion, or the act by which it is ejected from the body or organ in which it is formed, *e.g.* the lochial discharge (see PUERPERIUM, PHYSIOLOGY, *Lochia*), or the escape of accumulated energy, *e.g.* nervous energy in normal cerebral actions or in epileptic fits (see PHYSIOLOGY, NERVOUS SYSTEM, *Cerebrum*, *Durchgang Mechanismus*)

**Disclision.**—The incising or cutting into of the capsule of the lens in cataract operations, or the division of the cervix uteri from within outwards

**Disclination.** See CONJUNCTION

**Discrete.**—When the spots or pustules of a skin disease, *e.g.* smallpox, are separate and not coalescent, they are called discrete (*discretere*, to separate) See SMALLPOX (*Clinical Varieties*, *Variceloid*)

**Discromatopsia.** See DYSCHROMATOPSIA

**Discus.**—A disc, especially the discus proligerus of the Graafian follicles of the ovary, the cumulus proligerus or discus vitellinus See GENERATION, FEMALE ORGANS OF (*Ovaries*, *Microscopic Appearance*)

**Discussants.**—Medicines, applications, or methods of treatment having as their object the dissipation, dispersion, or resolution of swellings, effusions, tumours, or congestions, resolvers, commonly employed discussants are fomentations, friction, blisters, iodine, and mercury

**Disease.**—A disturbed, disordered, or deranged state of the bodily functions, due generally to structural alterations in some or all of the organs or tissues (*structural diseases*), but caused sometimes by conditions accompanied by no appreciable (or yet appreciated) lesions (*functional diseases*), diseases are also classified as congenital, constitutional, acute, chronic, contagious, idiopathic, general, local, nervous, organic, zymotic, etc. Sometimes the name of the medical man who first described a malady is given to it, *e.g.*—*Addison's Disease*, *Alibert's* (Fungoid Mycosis), *Aran-Duchenne's* (Progressive Muscular Atrophy), *Balfour's* (Chloroma), *Banti's* (Splemic Anæmia), *Basedow's* (Exophthalmic Goitre), *Bruan's* (Buccal Psoriasis), *Beard's* (Neurasthenia), *Bergbie's* (Exophthalmia), *Beard's* (Trichorrhæxis Nodosa), *Bell's* (Typhomania), *Bell's* (Ulceration of Lips), *Bergeron's* (Chorea), *Bright's* (Nephritis), *Buhl's* (Fatty Degeneration of New-born Infant), *Carrion's*

(Verruga Peruviana), *Charcot's* (Amyotrophic Lateral Sclerosis), *Concato's* (Polyorrhæmatis), *Chronic Peitonitis*, *Corrigan's* (Aortic Incompetence), *Cotugno's* (Sciatica), *Cruveilhier's* (Gastric Ulcer or Progressive Muscular Atrophy), *Darier's* (Acne), *Dercum's* (Adiposis Dolorosa), *Devergie's* (Lichen Ruber), *Diessler's* (Paroxysmal Hæmoglobinuria), *Dubini's* (Electric Chorea), *Duchenne's* (Pseudohypertrophic Paralysis), *Dühring's* (Dermatitis Herpetiformis), *Eichstedt's* (Dermatomycosis Furfuracea), *Fede's* (Sublingual Fibroma), *Flatau's* (Exophthalmic Goitre), *Fothergill's* (Trigeminal Neuralgia), *Friedreich's* (Hereditary Ataxia or Paramyoclonus Multiplex), *Fisher's* (Endemic Paralytic Vertigo), *Gilles de la Tourette's* (Impulsive Tic), *Giovannini's* (Nodular Disease of Hair), *Glénard's* (Enteroptosis), *Grancher's* (Spleno-pneumonia), *Graves's* (Exophthalmic Goitre), *Gull's* (Myxœdema of Adults), *Hammond's* (Aethetosis), *Hano's* (Hypertrophic Hepatic Cirrhosis with Jaundice), *Harley's* (Paroxysmal Hæmoglobinuria), *Hebra's* (Erythema Exsudativum Multiforme), *Henoch's* (Nervous Purpura with Colic), *Hirschsprung's* (Dilatation of Colon, Megacolon), *Hodgkin's* (Pseudoleucocythæmia), *Hodan's* (Variety of Trichorrhexis Nodosa), *Huguen's* (Uterine Fibroids), *Huntington's* (Chronic Chorea), *Kähler's* (Multiple Myelomatoma), *Kapov's* (Xeroderma Pigmentosum), *Krischke's* (Vertigo with Sensory Illusions, Cardiac Irritability, etc.), *Korakoff's* (Polyneuritic Psychosis), *Landy's* (Acute Ascending Paralysis), *Leber's* (Hereditary Optic Atrophy), *Little's* (Congenital Muscular Rigidity, Spastic Paralysis of Infants), *Mahli's* (Perivaginitis Simplex), *Milasse's* (Cystic Testicle), *Marie's* (Aciomegaly), *Ménier's* (Auditory Vertigo), *Mikulic's* (Enlargement with Sclerosis of Lacrymal and Salivary Glands), *Morton's* (Metatarsalgia), *Morvan's* (Variety of Syngomychia or Leprosy), *Munchinger's* (Progressive Ossifying Polymyositis), *Oster's* (Chronic Cyanosis with Polycythæmia and Enlarged Spleen), *Payel's* (Osteitis Deformans), *Payel's disease of Nipple* (Cancer (?) of Nipple), *Parkinson's* (Paralysis Agitans), *Parron's* (Syphilitic Pseudoparalysis), *Pavy's* (Exophthalmic Goitre), *Pavy's* (Intermittent Albuminuria), *Pott's* (Spinal Curves), *Quincke's* (Angioneurotic Oedema), *Ragnard's* (Local Asphyxia with Symmetrical Gangrene), *Recklinghausen's, von* (Generalised Neurofibromatosis), *Reclus's* (Cystic Disease of Mamma), *Reichmann's* (Nervous Dyspepsia with Supersecretion), *Riga's* (Sublingual Fibroma), *Ritter's* (Dermatitis Exfoliativa Neonatorum), *Rivolta's* (Actinomycosis), *Sachs's* (Amaurotic Family Idiocy), *Savill's* (Epidemic Eczema), *Schönlein's* (Purpura Rheumatica), *Stokes-Adams's* (Angina Pectoris with Bradycardia or Paroxysmal Bradycardia), *Sydenham's* (Chorea), *Tulma's* (Myotonia Acquisita), *Thomsen's* (Myotonia), *Thornwaldt's* (Cystic Disease of Pharyngeal Tonsil), *Tomaselli's* (Quinine Fever), *Wardrop's* (Malignant Onychia)

*Weil's* (Epidemic Catarrhal Jaundice), *Werthof's* (Purpura Hemorrhagica), *Whytt's* (Hydrocephalus), *Wilson's* (Generalised Exfoliative Dermatitis), *Winkel's* (Epidemic Hemoglobinuria Neonatorum), *Woolley's* (Primary Congestion of the Lungs)

## Disinfection.

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### I WAYS AND MEANS OF INFECTION

(A) *Ways of Ingress*—Infectious diseases may be communicated by inoculation, by contact or association with the sick, by vehicles, as excreta or water, etc, or (clothing and other articles contaminated therewith, and containing or conveying the microbes or their spores, by the bites, etc, of insects, and by residence in infected localities

The modes of ingress of the microbes into the fluids of the body may be distinguished as (1) inoculation, (2) absorption, (3) inhalation, and (4) ingestion. Some diseases are received by one, others by two or more of these means, and that with equal or unequal frequency. Inoculation and absorption being closely allied, a poison certainly inoculable is probably capable of absorption by an unbroken mucous surface.

1 *Inoculation*—The contagia of rabies (as hydrophobia), glanders, anthrax, and vaccinia, the extra-corporeal contagia of tetanus, erysipelas, and septicæmia, and the intra-corporeal leprosy are usually thus received, while an abrasion greatly facilitates the infection of syphilis, chancre, and gonorrhœa. Under exceptional circumstances others may be inoculated, as smallpox, diphtheria, and tuberculosis.

2 *Absorption* by mucous surfaces is the rule with venereal diseases and with puerperal septicæmia. Ophthalmia is the infection of the conjunctiva with the purulent discharge from other cases, in glanders and diphtheria the

microbes are arrested on the nasal, pharyngeal, or laryngeal mucous surfaces, and the bronchi rarely involved, except secondarily by extension. Diphtheria, measles, scarlatina, etc, are easily absorbed from infected pocket-handkerchiefs and like articles.

3. *Inhalation* is, however, the most frequent means of communication of all the non-recurrent intra-corporeal contagia, viz variola and varicella, typhus and plague, measles and rubella (rutheln), whooping-cough and mumps, as well as of yellow and relapsing fever and some others, also of tuberculosis of the lungs and cervical glands, contagious pneumonia, epidemic cerebro-spinal meningitis, influenza, and diphtheria. Enteric fever is, though rarely, and cholera may possibly be, thus received, and it has hitherto been universally believed that malarial fevers are conveyed aerially, though mosquitoes are now held to be the principal means.

4 *Ingestion* is certainly the rule with enteric fever, cholera, dysentery, and some forms of diarrhoea, water or foods specifically contaminated being the cause of epidemic outbreaks, and eating with hands soiled with feces, or the personal communication of the disease. The milk of tuberculous cows is the chief cause of infantile tuberculosis, intestinal, meningial, vertebral, etc. Foot-and-mouth disease of cows is communicated to man by their milk. Scarlatina, enteric fever, and diphtheria may also be conveyed by milk as a vehicle, there being no better culture fluid for most bacteria. Lastly, the imperfectly cooked flesh of tuberculous animals may be a means of infection.

5 *Insects*—Flies play a more important part in conveying infection than is commonly supposed. They carry infective matter on their feet and trunks from the sick to the sound and to articles of food, while quite recently a mass of evidence has been accumulating to prove that mosquitoes, i.e. gnats, of certain species, are the actual vehicles of malarial poisoning, the plasmodia or hamatozoa of these fevers swarming in the fluids of their bodies, and being inoculated by their bites into the human blood. Dogs and cats may convey infection in their coats, as human beings in their clothes, but cats are themselves susceptible of diphtheria, which they may thus communicate direct from one child to another.

(B) *Ways of Egress*—All pathogenic microbes leave the bodies of the sick by one or more ways, the chief of which are (a) the mucus secreted by the respiratory passages, throat, and mouth, (b) the intestinal evacuations; (c) the urine, and (d) the serous and purulent contents of vesicles and pustules, and possibly detached epithelium. The first-named is specially important in relation to scarlet fever and measles, and is probably the main source of the infection which not infrequently occurs

during convalescence of a case. Too exclusive attention has been attached to "desquamation" as the principal cause, to the neglect of the mucous membrane involved in the early and latest stages of the disease. The "breath" of patients suffering from infectious diseases is by some considered to be a means of infection, but the evidence for this is not conclusive. These points are of great practical importance in connection with the science and practice of disinfection.

Streptococci are present in large numbers in the urine of scarlatinal patients, but attempts to obtain cultivations from the cast-off epithelium have been so unsuccessful as to cast doubts on the universal belief in their infectivity, but Dr. Glass of Chicago states that he has grown from the mucus of the throat and the epithelium alike a diplococcus perfectly characteristic, and doubtlessly specific. He explains previous failures by the fact that the only media in which it will grow are glycerine agar with 5 per cent of sterilised garden soil, and milk.

(C) *Periods of Incubation and Infectiveness*.—A knowledge of the incubation or period that elapses between the reception of the infection and the first manifestation of the symptoms of the disease, and of that during which the convalescent retains the power of communicating the infection to others, is necessary to the successful practice of disinfection and prophylaxis, especially in schools.

*Incubation*.—The statement of these periods in our text-books is far from satisfactory, for they are, I believe, more constant than is generally supposed. An error on the side of excessive caution is certainly safe, but if we admit such wide ranges as two to fifteen days, or one to three weeks, we deprive ourselves of the means of tracing any case to its source.

*Deferred Infection*.—Many alleged instances of extraordinarily prolonged infection are really cases of recent exposure to infected articles, but it is also highly probable that one may carry the contagion for some days or a week on one's clothes or person, resisting infection for that time. For this Dr. Hencke has suggested the term "deferred infection," and such apparent prolongation of the incubation period is perhaps not infrequent.

*Incubation periods* should be reckoned from the moment of infection to that of the *invasion* or commencement of the febrile disturbance, not to the appearance of the eruption, which is a later stage in the course of some diseases, not having anything corresponding to it in others. The incubation is properly the period during which there is *no sensible evidence of disease*. Among the difficulties and sources of error in determining the duration of incubation are (1) the uncertainty attaching to the date of exposure alike when (a) no other cases are

known to exist in the neighbourhood, and (b) when, as during epidemics, opportunities of infection are everywhere present, (2) the possibility of deferred infection, or (3) of later infection by fomites, (4) the insidious course of some diseases, as enteric fever and diphtheria in their earlier stages, and (5) the fact that cases infected successively from the same source may be mistaken for primary and secondary cases with a very short interval between them.

Perhaps the following statement of the true periods may be taken as the nearest approach to accuracy.—

#### *Short Incubations*

Erysipelas	1-2 days
Septicæmia	1-2 "
Influenza	1-3 "
Diphtheria	1-4 "
Scarlatina	2-5 "

#### *Long Incubations*

Measles	10-12 days
Smallpox	10-15 "

#### *Variable Periods*

Cholera	1-4 days
Whooping-cough	7-10 or 12 "
Typhus	7-14 or 16 "
Enteric fever	12-20 "
Mumps	14-21 or 24 "

Varicella and rubella are occasionally somewhat shorter than smallpox and measles respectively. The eruption follows the invasion after one to two days in scarlatina, two to three in smallpox and typhus, and three to five in measles.

In a recent epidemic of rubella at Königsberg, Dr. Theodor and other medical men met a number of cases of the recurrence of the disease in the same individual after intervals of two to six weeks.

*Quarantine*.—A scholar who has been exposed to infection during the holidays should not be admitted, or, if in school, should be isolated for a period somewhat longer than the maximum incubation period, being meanwhile deemed a "suspect," and not allowed to associate with any susceptible individuals. This should be, in—

Scarlatina	8 days
Diphtheria	12 "
Measles	16 "
Smallpox	18 "
Whooping-cough	21 "
Mumps	24 "

Varicella and rubella as smallpox and measles.

*Isolation*.—*Duration of Infectivity*.—One who has suffered from an infectious disease should, after the most thorough disinfection of his person and clothes, be isolated from association with susceptible persons for the following periods.

*Scarlatina*—Eight or ten weeks from the appearance of the rash, desquamation having ceased, and so long after that as the throat is not perfectly healthy, or there is any discharge from the nose or ears.

*Measles and Rubella*—Three weeks from invasion, provided all cough, etc., have ceased.

*Mumps*—Four weeks, all swelling having disappeared.

*Whooping-cough*—Six weeks from the recognition of the cough, if it have entirely lost its spasmodic character, or four if all cough whatever have ceased.

*Diphtheria*—Four, or perhaps in very mild cases three weeks, provided there be no albuminuria or discharge from nose or ear, the patient be practically well, and the throat appear perfectly healthy.

A bacteriological examination of the pharyngeal mucus should be made on several consecutive days, for the bacilli may remain for some time virulent towards other persons, though inert towards the bearer.

*Smallpox*—When all scabs have fallen and the scars have healed.

In the case of the last two we have it in our power to protect susceptible persons from infection, viz vaccination or revaccination against smallpox, and prophylactic injections of antitoxin against diphtheria, the immunity conferred by these last persisting for about a month. The earliest commencement of infectivity is a question of some practical importance, especially in the suppression of epidemics in schools. There is no evidence of the communicability of any acute species during the purely incubative period, while as yet there are no objective symptoms, but it probably begins with the very earliest manifestation. All susceptible persons who have associated with the sick, on or subsequent to the first day of the invasion, should be suspects and put in quarantine, though when the disease is one with a long incubation, the isolation need not be enforced until the week following exposure. The infectivity is, however, but feeble, and prompt separation is, as a rule, successful, except with measles, in which infection mostly takes place before its nature is suspected. The evacuations of enteric fever and of cholera are infective when only a slight looseness is observed.

## II DISINFECTION AND DISINFECTANTS

*Disinfection*—(General practical disinfecting processes are directed to—(1) the destruction of all microbes deposited with the dust on all surfaces and projections, and in crevices and recesses on or in the walls, floors, ceilings, furniture, etc., of rooms, or adhering to or contained in furniture, carpets, curtains, decorations, etc., of rooms, as well as clothing and other domestic articles, (2) the destruction of the infectivity of excreta and expectoration, (3)

preventing the spread of infection by persons, and (4) destroying *in situ* such microbes as, though pathogenic, have also an extra-corporeal or saprophytic existence.

The removal of offensive odours and the prevention of putrefaction have a certain and occasional value, but there can be no greater error than the popular confusion of deodorants, and even of antiseptics, with disinfectants—that is to say, with germicides—although some bodies partake of the properties of more than one of these groups.

*Deodorants* that simply overpower an ill odour by substituting an agreeable one are worse than useless, those only having any real value that break up offensive and injurious volatile bodies, as hydrogen or ammonium sulphides, and combining with some or all of their molecules, fix them in inert and inodorous compounds, and those which, giving off nascent oxygen to organic matter in a state of incipient decomposition or unstable equilibrium, oxidise it with such rapidity that the process has been aptly termed “wet combustion.” These are represented respectively by the actions of chlorine, and of permanganate of potash and peroxide of hydrogen. The latter, being mere oxidisers, have little action on living bacteria. They have been fully discussed in the article “Antiseptic Treatment of Wounds” in vol. i p. 299.

*Disinfectants*, in the restricted sense that the word has now acquired, are *germicides*, i.e. bodies capable of killing bacteria, and, in practice, such only as do so *effectually* and *permanently*, for one that does not, fails altogether to fulfil its purpose.

Then absolute and relative values must be learnt by laboratory experiments conducted on strict scientific methods, but in applying the conclusions thus arrived at to actual practice one must take account of the different conditions under which they are employed, and of the disturbing factors which, rightly excluded from the laboratory, have to be reckoned with elsewhere.

It is not enough to know that a substance is a germicide, one must know and must use it in the requisite quantities and degree of concentration, nay more, one must make sure that it comes into actual contact with the whole of the bacteria. Very few indeed of so-called disinfectants fulfil all these conditions, and if they fail in one, they are but illusions and shams, little if at all better than the perfumery fumigation of travellers and their luggage that until recently satisfied the officials in some countries. Thus the 1 lb of sulphur, or 11 cubic feet of sulphurous acid gas per 1000 cubic feet of space, now prescribed for the disinfection of rooms, is based on Koch's observation that an atmosphere containing 1 per cent of the gas was fatal to all bacteria in thirty minutes, though not to their spores.

But Koch recognised the difference between his empty air-tight box and a furnished room with leakage by doors and windows, and showed that even in his experimental chamber similar cultures covered by a cloth or in a few folds of filtering paper were protected from the action of the gas.

Again, medical men who clearly appreciate the uses and action of sublimate solutions of 1 in 1000 or in 2000 respectively, are found advising the addition of indefinite quantities of the reagent to still more indefinite volumes of excreta or sewage, by dilution with which the proportion of the sublimate would be instantly reduced to anything between 1 in 10,000 and in infinity, whereas the quantity of the reagent to be used should be calculated on the liquid to be disinfected, as by adding 2 oz. of a 1 per cent solution to a pint, or 16 oz. to a gallon of the excreta, if a strength of 1 in 1000 is desired.

Even then the fluid mixture should be agitated, and, if thick, be diluted before treatment, for the dense coagula formed by the action of the sublimate on albuminoid matter may otherwise enclose and protect the bacteria, so that disinfection is incomplete.

The insufficiency of sulphur fumigations as commonly performed may be demonstrated by placing one silver coin on a table and another in the pocket of a coat during the process, when the former will be found blackened and the latter scarcely tarnished.

**Heat as a Disinfectant.**—Heat is the most certain means of disinfection. Our exact knowledge of its efficacy dates from the investigations of Koch, Wolfiugel, Gaffky, and Loeffler in 1881, whose conclusions were that—

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But few fabrics, if any, can stand long exposure to such temperatures in dry air without serious damage, and several hours are required for the heat to penetrate the interior of mattresses or bales of goods. Moist heat is, however, far more effectual, and does not injure the majority of fabrics, leather being the most important exception, while exposure to saturated steam at  $100^{\circ}\text{C}$  for 15 minutes suffices to kill even such resistant organisms as the *B anthracis* and its spores. Whether steam is more energetic at high pressures, as 20 lbs. to the square inch, than at lower pressure is, though probable, not determined, but its power of penetration is undoubtedly greater, and consequently a shorter

exposure is necessary, and there is less condensation of moisture on the articles submitted to it. An incidental advantage is found in the fact that where steam power is employed for other purposes it is always worked at high pressure, and may be utilised for the disinfecting apparatus, though the initial cost of the stronger chamber required will be greater. The penetration of the steam may be accelerated by intermittent raising and lowering of the pressure, the steam being let off, and after some minutes turned on again. The removal of the moisture of condensation is facilitated by producing a partial vacuum, and then admitting dry hot air at atmospheric pressure. The exhaustion is best effected by passing a jet of steam across the mouth of a pipe communicating with the chamber until the gauge indicates a "vacuum" of 20 inches, when air is admitted at atmospheric pressure by a pipe surrounded by a steam coil that raises its temperature to  $105^{\circ}\text{C}$ , and the creation of a "vacuum" previously to the admission of the steam is, as regards its penetration, equivalent to raising its pressure, and permits of equally rapid disinfection at lower temperatures, with consequent less risk of injury to the goods, a temperature of  $105^{\circ}\text{C}$  being under these circumstances as effective as one of  $120^{\circ}\text{C}$  would be otherwise.

Washington Lyon's apparatus is perhaps the best, as it is the most costly. Reck's fixed and portable are excellent, and much less expensive; and Thresh's and Defies' "Equivex" have each features to recommend them.

**Chemical Disinfectants.**—Innumerable inorganic and organic bodies possess more or less of germicidal properties, and numbers of these, alone or in combination, have been put on the market as proprietary preparations, but too often of unknown and very uncertain composition, some being good, though their value be exaggerated, while others are feeble almost to inertness.

Chemical disinfectants may be considered under the several heads of (1) oxidisers, bodies giving off oxygen in the nascent or atomic state, and therefore very active, (2) halogens, or bodies evolving chlorine, bromine, or iodine in the nascent state, (3) oxides of nitrogen, (4) caustic alkalis, (5) acids, (6) metallic salts, (7) the phenols and their derivatives, and (8) formic aldehyd.

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In "sanitas," however, prepared by passing air through oils of turpentine, camphor, etc., floated on water, there is much  $\text{H}_2\text{O}_2$  and some  $\text{O}$ , formed in the slow oxidation of the "essential" oils, and potassium permanganate, in the presence of acids or of organic matter, especially



if in a state of nascent decomposition, gives off "nascent" oxygen freely. The latter, commonly known as "Condy's fluid," is largely used for washing foul wounds, removing the smell from the hands or from vessels that have been in contact with putrid or fecal matters, and for sweetening meat that is slightly "tuned" or game already too "high." It is, however, but a feeble germicide in solutions of less than 5 per cent, and unless very dilute, it stains fabrics a deep brown. Sanitas in 2 per cent solutions may be used for the same purposes, and is free from these defects, being non-poisonous, colourless, and non-irritant. It makes an elegant toilet preparation, and is a fairly active oxidiser.

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strength of these proprietary preparations is a great defect.

**Mercuric Salts.**—The cyanide and bimide are now preferred in surgery, but the bichloride  $\text{Hg}_2\text{Cl}_2$ , commonly called corrosive sublimate or simply sublimate, stands unrivalled as the universal disinfectant for its energy, general applicability, and cheapness. Its poisonous character is often urged against it, but the danger is far less than is commonly supposed, the smallest fatal dose being 3 to 5 grains, or  $\frac{1}{4}$  to  $\frac{1}{2}$  pint of the 1 in 1000 solution, which is the strongest used. Such a quantity could not be drunk by accident, and the ounce that might be swallowed in mistake would represent  $\frac{1}{2}$  grain only, the worst effect of which would be a little gastro-intestinal disturbance, whereas the same quantity of Burnett's Fluid, or a half or even a quarter of an ounce of carbolic acid, would very probably prove fatal. Still, if people are afraid to have it in a house, it might be coloured blue with indigo or laundry blue, when it could not be mistaken for any medicine or beverage.

One part in 10,000 or 1 in 5000 is fatal to all but the most resistant bacteria, and 1 in 1000 suffices to kill in a few minutes even those of anthrax in water, and all others even in thick fluids or feces. To obtain this proportion a stock solution of 1 per cent may be kept, with 10 per cent of common salt or 0.1 per cent of hydrochloric acid added to prevent deterioration through the deposit of a basic chloride.

(7) **Phenol**,  $\text{C}_6\text{H}_5(\text{OH})$ .—Commonly called carbolic acid, it is rather of the nature of an alcohol or hydroxyl derivative of a hydrocarbon radical. It is strongly antiseptic, but neither powerful nor certain as a disinfectant, for unless concentrated and allowed to act for several days it does little more than delay the germination of spores. Koch found that 1 per cent destroyed the bacilli and 5 per cent the spores of anthrax in two days, but some bacilli are even more resistant, and those of typhoid fever flourish in a medium carbolised to 1 per cent.

Carbolic powders consist of phenol incorporated with inert mineral matter, its minute subdivision favouring its volatilisation, a doubtful advantage since actual disinfection is an illusion. They should be guaranteed to contain 15 per cent of phenol, though few do, and some show only a trace.

The crude acid of the shops is in some respects superior to the pure.

**Cresol** or **methyl phenol** with its derivatives forms the chief constituent of lysol, cresolin, izal, "Jeyes' Fluid," and a host of similar mixtures and preparations. It is a better disinfectant than phenol, and some of its products and derivatives are much less poisonous.

(8) **Formaldehyde**,  $\text{COH}_2$ , **Paraformaldehyde**,  $\text{C}_6\text{O}_3\text{H}_4$ ,  $\text{H}_2$ .—Polymers of the aldehyde of methyl, the first being a pungent gas, very soluble in

water, from which it is given off again on heating, and the second a white crystalline solid, breaking up when heated into the normal aldehyde. It is a very powerful antiseptic, recently much used for preserving milk, fish, etc., and a very good disinfectant. In 1 per cent solutions it kills all microbes in from fifteen minutes to one hour, and not being caustic or irritant, and not forming an insoluble compound with soap, as sublimate and zinc salts do, it may be used without hesitation for disinfecting the hands, clothing, brushes, etc., and in laundry work. It has a decided action on bacteria even as a dry gas, though far less than when in solution or when the articles have been wetted. The so-called "Formalin" is a 40 per cent solution of formaldehyde, and paraformaldehyde is sold in tablets, to be volatilised in a special lamp, the "Alformant." When this is used the walls and furniture should be previously sprayed with water. It may be described as *the domestic*, as sublimate is, or should be, *the official* disinfectant.

Disinfecting soaps and candles are not to be commended, being too feeble to be of any real use, while giving a false sense of security.

### III.—PRACTICAL DISINFECTION

**Preparatory Measures.**—The difficulties of disinfection, the risk of spoiling things in the process, and the necessity of destroying such as cannot be thoroughly disinfected would be minimised if the moment an infectious disease is recognised, or even suspected, all carpets, woollen curtains, stuffed furniture, feather beds, down quilts, and rugs were removed from the room, as well as clothing in chests of drawers, wardrobes, or hanging closets, and no furniture retained beyond a table and cane or wooden chairs, washing-stand, and the like. The easy American or Indian chairs, into the construction of which no textile fabric except a little canvas enters, should be substituted for the arm-chairs or couches usually provided for the attendants. The oldest and the least valuable blankets and bedding should be brought into requisition, or, better still, the ordinary mattress and flock or hair bed be exchanged for one stuffed with the cheap but comfortable zosteria or sea-wrack, commonly though incorrectly called "alva," which may afterwards be burnt, the ticks being boiled for future use. As at this early stage infection can scarcely have taken place, it will be sufficient to expose the articles removed to the wind and light in the garden or yard.

A room on the highest floor, if lofty and spacious with ample windows, is to be preferred, but on the next below, if the topmost room be in the roof with low ceilings, dormer windows, and the like, the door should be kept closed and the window more or less open, and if the weather permit, a fire, however small, kept burning, if not, the chimney at any rate should

never be closed. All windows on the staircase should be open day and night, and the doors and windows of other bedrooms during the day, as should the door into the garden, if any, and the front door also if kept on the chain.

*Special Considerations*.—These anticipatory precautions may with advantage be taken in all cases of known or suspected infectious disease, but when its character is determined the subsequent steps for preventing the spread of the infection will be regulated as to both their nature and the stringency with which they shall be enforced by the circumstances of the case.

The medical man will have to take into account (1) the presence of susceptible persons, especially with regard to the age-incidence, and fatality of the disease, (2) its degree of communicability to susceptible persons and the danger to life involved, (3) the persistence or vitality of the microbes and their spores out of the body, (4) the "ways and means" of infection.

Thus (1) all persons who have not previously passed through an attack of scarlatina are susceptible, but the susceptibility and fatality is so far greater in childhood than adults over 20 or 30 years of age need not be taken much account of, and those past middle life may be looked on as insusceptible. (2) Measles is the most infectious of all diseases (except smallpox in an unvaccinated community), but the danger to life, except in infancy, is practically nil, while the fatality of diphtheria at all ages is greater than that of almost any other. (3) The infection of measles is evanescent, while those of scarlatina and of diphtheria are persistent to an extraordinary degree. (4) Enteric fever is spread almost exclusively by the *feces* and *urine*, which, gaining access to water-supplies even after percolating many yards through the earth, may infect an entire community, though in a spacious and well-ordered sick-room the danger to the attendants and household is insignificant. On the other hand, the infection of scarlatina, diphtheria, or smallpox is with ordinary care easily confined to the house, though within it susceptible persons are with difficulty kept from contracting it. But, *pro contra*, no one need be susceptible, at least after the first few days, to smallpox or diphtheria, for in revaccination we have the means of conferring immunity for many years, and prophylactic inoculation with diphtheritic antitoxin protects from infection for about a month, measures that should never be omitted on the appearance of either disease in a house.

*Economy of Sick-room*.—During the course of the illness all linen when soiled or changed should be immediately immersed in a solution of "formalin," 1 part to 20 of water, or sublimate 1 in 2000, or zinc chloride 1 in 25, in a glazed earthenware pan, such as is used for

keeping bread, and as soon as convenient they should be plunged for a quarter of an hour in a copper of boiling water. Before applying soap to the clothes it will be necessary to rinse and wring them out in clean water if sublimate or the zinc salt have been used, since these, especially the latter, form insoluble compounds with the fatty acids. With "formalin," which does not in the least interfere with the lather, this precaution is not needed, and a wooden or galvanised tub may be used. Cups, plates, spoons, forks, etc., for the use of the patient should be kept and washed in the room, and on no account sent downstairs.

All *evacuations* should be immediately well stirred with a 1 per cent solution of sublimate in the proportion of 2 oz. to the pint before being passed into the sewer or buried in the earth, unless received for the inspection of the medical attendants in the open air, not in a closet or in a room. The urine is *at least* as infective as the feces.

*The Attendants*.—Persons in attendance on a patient suffering from smallpox, diphtheria, or scarlatina, and, of course, typhus—though this is not likely to be met with in private practice—should avoid all contact with susceptible individuals, especially children. If this be impracticable, as when the circumstances of the family preclude the engagement of professional nurses, and the wife or mother must take her turn with the patient and the other children, she should, as indeed should all nurses, wear only cotton dresses, and these changed twice a week at the least, while one that has been soiled by the excreta in enteric fever, or by the nasal and oral discharges in diphtheria or scarlatina, should be at once consigned to the disinfecting tub. She should have another dress hanging on the landing to put on in the house, leaving that she had been wearing behind in the room.

She must keep her nails short, and after dipping her hands in the formalin solution (1 in 20), cleanse them with hot water and soap, using the nail-brush, before attending to the other children.

It would be most desirable that her hair should be cut short enough to be frequently washed, but if not it should be enclosed in a cotton cap while in the room, or a bathing-cap would be still better.

*Convalescents*.—After smallpox and scarlatina, so soon as recovery has well advanced, the person of the patient should be disinfected by frequent baths with hot water and soap, the hair cut short and the head washed with formalin followed by soap and water, soft soap being preferable to hard. The throat in scarlatina and diphtheria should be sprayed with a weak solution of Liq. sod. chlorinate or formalin or Liq. pot. perm. daily for several weeks, or so long as there is any redness, swelling, or relaxation. The use

of a spray and not a gaigle is to be recommended. Dr. Sanger's colour experiments prove conclusively that a gargle does not come in contact with the walls of the pharynx or the tonsils, and can act on the soft palate and arch of the fauces only.

When convalescence is complete, and the danger of infection considered past, it is advisable that the patient, before returning to his family, should, especially if he have been in a hospital or in a room with other patients, be sent away for a week or two where he may breathe a pure air and eliminate the last traces of infection.

Attendants who, though insusceptible to scarlatina, or not having contracted diphtheria, are sensible of some degree of sore throat, indicating resisted and abortive infection, should use the spray to their throats and pass a week or so in a pure air, as much as possible out of doors, in the same manner, to rid themselves of the germs which, though they have had little effect on them, may communicate the disease to others more susceptible.

*Special Considerations*—As with the precautions to be taken to prevent the spread of infection during the illness of the patient, so with the subsequent disinfection of the room, bedding, etc., a certain latitude may be allowed according to the nature of the disease. With measles and whooping-cough, the microbes of which perish very soon, a thorough cleaning of the room, washing of linen, blankets, etc., and exposure of beds, pillows, and unwashable curtains, carpets, etc., such as is familiar to housekeepers as a "spring cleaning," suffices. With enteric fever no more is needed except for bedding and linen that have been in contact with the patient, but beds and mattresses should be destroyed rather than disinfected, since they are sure to be more or less saturated with the fluid and mostly involuntary evacuations of the patient. In puerperal fever, a septic disease, the contagion of which maintains an extra-corporeal existence, and is most persistent, the destruction of the bedding is imperative, for the neglect of this precaution may cause the death of a parturient woman occupying the bed even after the lapse of a year or longer.

In diphtheria, scarlatina, and smallpox it is highly advisable to substitute for pocket handkerchiefs pieces of soft cotton or linen rags, which should be burnt as often as used, or the Japanese paper handkerchiefs if they are to be had.

*After Death*—Infection does not cease with death. The number of well-authenticated instances of the propagation, sometimes widespread, of smallpox, diphtheria, scarlatina, typhus, etc., through contact, direct or indirect, with the bodies of persons dying of those diseases is so great that much more stringent legislation would be a public benefit. At any rate, the body should, so soon as possible, be put into the

coffin, bedded in some disinfecting absorbent, the best being probably Hartmann's sublimated wood-wool, or, in rougher practice, sublimated sawdust, "formalin" might be sprayed over all. The coffin should be placed in an empty room, covered with its lid, though so soon as the first signs of cadaveric change appear it should be screwed down, and burial (or cremation) follow at the earliest possible date. There is no excuse for "last looks," and to allow a "last kiss" is morally criminal. When an empty room is not available, or for other reasons the medical attendant deems the retention of the corpse a danger to the health of the inmates of a house, he can obtain an order from a magistrate for its removal to the mortuary, and its burial from thence, and it is much to be desired that public mortuaries were largely and voluntarily used for the deposit of corpses between death and burial, irrespective of social position, of accommodation, or the nature of the disease.

The practice in Munich and the arrangements of the magnificent mortuary of that city, where rich and poor alike "lie in state" and surroundings suggestive rather of a sacred edifice than of the dead-house and post-mortem room, well deserve study and imitation. It bears no resemblance to that "chamber of horrors" the Morgue at Paris, the purpose of which, to preserve unclaimed bodies as long as possible, is entirely different.

*Disinfection of Room, etc.*—The common method of aerial disinfection by means of sulphur is very inadequate. Whatever efficacy it appears to have is doubtless due to the thorough and prolonged influx of fresh air which follows its use. The object of disinfection is to kill the germs adhering to the walls and floors, accumulating in the dust deposited on ledges, cornices, and furniture, and lodging in the folds of woollen fabrics and the stuffing of bedding, chairs, etc., just the places where they are least accessible to gaseous agents.

The first step is to have all washable fabrics plunged in boiling water for a quarter of an hour or twenty minutes, and mattresses, beds, bolsters, pillows, and, if such have unfortunately been left in the room, all quilts, carpets, rugs, and the stuffing of easy-chairs, packed up for transmission to the disinfecting station. Next, the floor should be well washed, in fact swilled, with 1 in 1000 sublimate solution, care being taken to saturate the interspaces between the boards, and the walls sprayed with the same by means of a garden syringe or other apparatus, or simply washed down with a cloth or mop. The ledges over the door and window frames and the wood-work of the sashes should be washed with a cloth dipped in the solution, and the furniture treated in the same way. The ceiling should then be limewashed, special care being paid to ornamental cornices, roses, and other decorative

work, that no part shall escape the application. The room should then be left with the windows open for a day or two, after which the ceiling may be whitewashed or papered, the walls, if papered, stripped and repapered, and the floor scrubbed with soap and soda, which will convert any remaining sublimate into a non-volatile and insoluble compound. A fresh coat of paint to doors and windows would be a further security. Fenders, fire-irons, and metal work generally, which would be spoiled by the sublimate, need only be well polished.

Where a proper steam disinfecting oven is available, all beds, carpets, and such-like should be sent there, or if there be none available the ticking and covers must be boiled, good hair or feathers may be steeped in a 1 in 1000 sublimate solution for half an hour and then washed in pure water, but flock or cheap hair and mixed stuffings are far better burnt.

**Legislation.**—The various enactments bearing on the prevention and repression of infectious diseases will be referred to in the articles INFECTION, QUARANTINE, etc.

**Dislocation.**—The separation or displacement of bones (more especially) from their natural relations to each other. It may be complete (the ends of the bones overlapping) or incomplete (partial), simple (no other injury), or complicated (one or both bones fractured), or compound (wound making the joint communicate with the exterior), habitual or relapsing (recurrent), spontaneous (not due to violence), or traumatic (due to violence). See ANKLE-JOINT, REGION OF, INJURIES (*Dislocation of Peroneal Tendons, Tibio-Tarsal and Compound Dislocations, and Dislocation of Astragalus*), BRACHIAL PLEURA, SURGICAL AFFECTIONS OF, CHEST, INJURIES OF (*Dislocation of Ribs*), DEFORMITIES (*Congenital Dislocations*), ELBOW-JOINT, INJURIES AND DISEASES OF (*Dislocations*), FINGERS (*Injuries, Dislocations*), HIP-JOINT, INJURIES OF (*Dislocations*), KNEE-JOINT, INJURIES OF (*Dislocations*), LENS, CRYSTALLINE (*Displacement*), MOUTH, INJURIES AND DISEASES OF THE JAW (*Dislocation of Lower Jaw*), NERVES, PERIPHERAL (*Dislocation of Ulnar Nerve*), SHOULDER, DISEASES AND INJURIES OF (*Dislocations of Humerus, Acromial End of Clavicle, and Biceps Tendon*), SPINE, SURGICAL AFFECTIONS OF (*Fracture-Dislocation*), STERNO-CLAVICULAR JOINT (*Injuries*), WRIST-JOINT, INJURIES (*Dislocations*).

**Disomata.**—Double monsters or united twins, in contrast to the monosomatosus terata or single monsters (*Tamifii*). See TERATOLOGY.

**Disorder.** See DISEASE.—Disorder is generally regarded as a milder term than disease, and does not indicate structural alterations.

**Dispar.**—Unequal, unlike.

**Dispensary.** In the strict sense dispensary means a place where medicines are made up or dispensed, but it has come to mean also an institution where poor patients are seen, examined, prescribed for, and given medicines and (sometimes) surgical appliances, gratis, or for a nominal fee (Charity or Public Dispensaries).

**Dispensatory.**—A non-official Pharmacopœia, containing, in particular, the pharmaceutical details respecting drugs.

**Dispensing.**—Making up or putting up medicines according to a prescribed formula (i.e. a prescription). The dispenser has to be very careful to read the prescription carefully and thoughtfully, to be alert to detect incompatibles or wrong doses, to label the medicine carefully ("Poison," "External Application only," "Shake the Bottle"), etc. See PRESCRIBING.

**Displacement.**—A dislocation, more especially of one or other of the internal organs, e.g. of the uterus, spleen, etc.

**Disposal.**—Disposal of sewage, of refuse, of the dead, etc. See SEWAGE AND DRAINAGE, etc.

**Disposition.**—Constitution or diathesis or tendency.

**Dissection - Wounds.** See POST-MORTEM METHODS (*Rules of Procedure, Personal*).

**Disseminated.**—Scattered or discrete, as in disseminated sclerosis. See PARALYSIS (*Paralysis with Tremor or Ataxy, Disseminated Sclerosis*).

**Dissociation.**—The separation and recognition of the elements of a tissue or organ by histological methods (staining, teasing, etc.). Dissociation of personality means the breaking up of the "Ego" into two or more "sub-conscious partners," as in Professor Prince's patient, "Miss Beauchamp."

**Dissolution.**—The breaking up of a tissue or of the anatomical elements of a tissue, morbid softening of a tissue, death, or the disappearance of all traces of an embryo which has died in early antenatal life.

**Dissolution, Law of.**—Drugs (e.g. alcohol) acting on the brain and spinal cord are subject to what has been called the Law of Dissolution, "when a drug affects functions progressively, those first affected are the highest in development—that is to say, they are the last acquired by the individual and the last to appear in the species. The next affected are those next to highest, and so on, till finally the lowest of all from an evolutionary point of view, that is to say the functions of respiration and circulation, are affected" (Hale White). See ALKALOIDS (*Law of Dissolution*).

**Distal.**—Distant or remote, opposed, therefore, to proximal, the distal end of a long bone is that farthest from the trunk. *Distad* means in the direction of or towards the distal end of a bone or a limb, etc

**Distemper.**—An infectious catarrhal disease affecting carnivorous animals, especially dogs, and consisting in inflammation of the mucous membrane of the nose, throat, eyes, bronchi, and alimentary tract, the skin is sometimes affected, it is most common in dogs under one year of age and is very fatal (about 50 per cent), it is a sort of "dog-measles," and one attack usually confers immunity. The word *distemper* may also be used of any disease.

**Distichiasis.**—The presence of a second row of eyelashes, occurring as a congenital anomaly. See EYELIDS, AFFECTIONS OF (*Distichiasis*).

**Distoma or Distomum.**—The *distomae* belong to the trematodes or flukes among the parasitic worms, there are several species including *distomum hepaticum* (the liver fluke), *distomum lanceolatum* (the smaller liver fluke), *distomum hematobium* (bilharzia hematobia), etc. See LIVER (Liver Parasites), LUNGS, PARASITIC AFFECTIONS OF (*Distomum Ringeri*), PARASITES (*Helminths, Trematodes*).

**Distomus.**—The teratological state in which there is a double mouth or double lower jaw (Ballantyne's *Antenatal Pathology*, vol II pp 389, 447).

**Dita Bark.**—The dried bark of *Alstonia scholaris*, containing an alkaloid *ditaine* ( $C_{41}H_{30}N_2O$ ) having a paralyzing effect on motor nerve endings (in mammals), it is official in the Indian and Colonial Addendum (1900) to the British Pharmacopoeia of 1898. See ALSTONIA.

**Dittrich's Plugs.**—Yellowish plugs of sputum, varying in size from a millet seed to a bean, formed in the bronchi in cases of gangrene of the lung and bronchiectasis. See BRONCHI, BRONCHITIS (*Clinical Varieties, Purulent Bronchitis*), EXPECTORATION (*Form*).

**Diureides.**—Bodies consisting of two unmodified or modified urea molecules, linked together by an acid nucleus, e.g. the purin bodies, in birds and reptiles they are the substances in which nitrogen is principally eliminated, the most important of them is uric acid, and others are xanthin, hypoxanthin, and allantoin. See LIVER, PHYSIOLOGY OF (*Regulation of Supply of Proteids*), PHYSIOLOGY, EXCRETION (*Nitrogenous Substances, Diureides*).

**Diuresis.**—Increased or abundant excretion of urine, occurring as a sign of disease, as

the result of taking some medicines, or because of altered physiological conditions, polyuria. See DIURETICS.

**Diuretics.** See also ALCOHOL, BUCHU, CALOMEL, CAFFEINE, DIGITALIS, DIURETIN, HEART, MYOCARDIUM and ENDOCARDIUM (*Treatment, Strophanthus and other Cardiac Remedies*), JUNIPER, POTASH, SQUILL, etc, etc.—A diuretic is usually defined as an agent which increases the elimination of urine. Such a definition is a convenient clinical one, but we must bear in mind that in certain diseases, e.g. granular contracted kidney, the failure in excretion is one in the elimination of solids, and not in the discharge of water, and in such cases there are important indications in connection with the daily intake of nitrogenous ingredients.

The difficulties that invest this subject either from the pharmacological or clinical point of view are considerable. They depend largely on the fact that very considerable variations in the total excretion of urine occur not only in health, but also in many diseases,—variations which are apparently quite independent of the dietetic or other treatment in operation.

Thus in health we find variations occurring quite independent of the amount of fluid ingested, and also independent of the amount lost by the various other channels, these variations depending on the varying activity of the renal structures.

The mechanism of renal secretion will be fully discussed in the article "Kidney", here it will suffice to give a general outline of the factors which are concerned in diuresis, at the same time indicating the different points that call for consideration in seeking to estimate the potency of an agent with supposed diuretic influence.

As a proof of the very marked variations in the amount excreted from day to day, quite independently of the employment of any agent of supposed diuretic influence, the following figures may be quoted—

Case I		
June 8,	1200 c c	urine
" 9,	1352	"
" 10,	1700	"
" 11,	1305	"
" 12,	990	"
" 13,	1645	"
Case II		
Oct 29,	1865 c c	urine
" 30,	1950	"
" 31,	2375	"
Nov 4,	2470	"

These are taken from the middle of a series of detailed observations on the urine of two hospital patients under similar treatment from day to day, and one can readily imagine from



these figures that a powerful diuretic influence might be attached to a drug employed at a time when the renal secretion was low. The cause of these variations is unknown, but their existence must be recognised when seeking to define the diuretic action of any remedy in use.

There are many things which have to be carefully considered in connection with the subject of diuresis, and there are many points of difficulty in their consideration—points depending on our want of accurate knowledge of the agencies concerned in the renal secretion in health as well as disease.

The *force driving the blood* through the kidneys has first to be considered. This has in great measure to be determined by the state of the general blood-pressure as estimated by the pulse. We must, however, bear in mind that there may be increased vaso-motor tone in the kidney, of local origin, dependent upon alterations in the splanchnic area, and it is interesting in this connection to note the close relationship which has been shown by Hill to exist between the splanchnic area and the regulation of the cerebral circulation. As an instance of diuresis resulting from general increased arterial tension we have the influence of cold.

Then the *state of the blood* itself has to be considered. It may be that the variations in the amount of urine excreted from day to day in some cases of gout are largely dependent on the relative amount of toxic material present. Apart from the presence of toxic substances, excess of uric acid in the blood and the amount of salts require consideration, and will be referred to later. The *condition of the kidneys* themselves is a further point meriting very careful consideration. On the one hand there may be some defect in the state of the secreting structures in the glomeruli and the tubules, or some obstruction in the excretory channels, or, on the other hand, the permeability of the kidneys may be impaired, or there may be interference with the venous circulation. While all these factors have to be borne in mind, it is exceedingly difficult and frequently impossible to determine which of them has been at fault, or, in other words, which of them has been influenced, and how, by the diuretic used in any given case. In disease we may find a greater permeability associated with diminished velocity, and obstruction to the discharge from the kidney.

Diuresis may occur through an elevation of the arterial tension, through an excess of water, or by an excess of salts.

The ingestion of water probably acts in a complex manner: the total bulk of the blood is augmented, tissue metabolism is modified, with resulting increased excretion of waste products, and the renal capillary pressure is raised. An excess of salts acts similarly, but in different directions: here fluid is withdrawn from the

cells, thus tending to increased total bulk of that fluid; further, the osmotic pressure is increased, and the general metabolism as well as renal metabolism influenced.

It has been thought that one reason why potassium salts are more powerful diuretics than sodium salts depends on the fact that the former are naturally less abundant in the blood, and are therefore more stimulating and active on account of their greater diffusive power.

The action of alkaline and acid salts differs somewhat from those of the neutral, the former increase production of urea, which being a stimulant to renal excretion promotes diuresis. Acid salts tend to diminish urea production, but induce a flow of alkaline fluid from the tissues, and thus increase the amount of salts and water in the blood with resulting diuresis.

The compounds of the xanthin group, notably caffeine, act by modifying renal metabolism, and by also stimulating the circulation.

*Indications for the use of Diuretics.*—Diuretics are so frequently indicated in various cardiac, respiratory, and renal diseases, and also in many conditions of general disordered metabolism, that only a few general statements need be quoted.

The first essential for the clinician in all cases is to endeavour to gauge the state of the kidney itself and the state of the cardio-vascular system. This has to be done by a detailed general examination of the subjective and objective symptoms present in each case.

In all cases, moreover, it is advisable that the functions of the auxiliary excretory organs be judiciously promoted. The use of a vegetable purgative pill, or calomel in pill form, with an occasional morning saline, and the use of hydrotherapeutic remedies to promote the skin function, are very important aids to treatment.

If, after consideration, the conclusion is arrived at, that the defective secretion results from low vascular tension, digitalis, or a similarly acting body, is the remedy *par excellence*. A prescription like the following can be recommended—

R Potass. citrat	gr xxx
Spm chloroform	℥℥
Tinct digitalis	℥℥
Infus buchu	℥ss

Dose for an adult, *t d s*, to be followed by a copious drink of water.

The foregoing illustrates how we may advantageously combine diuretics of different classes, the digitalis acting mainly through the general blood-pressure, the potash and buchu acting on the kidney structure, directly aided, in the case of potash, by alterations in general tissue metabolism. (Tate of potash or lithia alone, or in combination with buchu or scoparium as a vehicle, may be mentioned as suitable drugs in cases where a vascular diuretic is not indicated.

The nitrites in large doses are also serviceable remedies, then exact mode of action being unknown. As previously mentioned, the diuretic action of plain water either alone or following the use of diuretic drugs cannot be too strongly emphasised. As a general rule it should be taken on an empty stomach at set times, three or four times daily, in quantities varying from one-half to one pint or more.

**Diuretin.**—A proprietary diuretic medicine, salicylate of theobromine and sodium, it is used in dropsy due to heart and kidney troubles, the dose is 5 to 15 grains.

**Divagation.**—Rambling speech or thought, especially of the insane.

**Divalent.**—*Divalent* or *bivalent*, as applied, for example, to an acid, means "capable of replacing two atoms of hydrogen in a compound."

**Divergence.** See OCULAR MUSCLES, AFFECTIONS OF (*Paralysis*).

**Diver's Paralysis.** See SPINE, SURGICAL AFFECTIONS OF (*Caisson Disease*).

**Diverticulitis.**—Inflammation of Meckel's diverticulum.

**Diverticulum.**—A side-branch of a canal or cavity, especially one ending blindly, a cul-de-sac. Some diverticula have special names, e.g. *Meckel's diverticulum* (a cul-de-sac arising from the lower part of the ileum), *Nuck's diverticulum* (canal of Nuck), *duodenal diverticulum* (ampulla of Vater), etc. See INTESTINES, SURGICAL AFFECTIONS OF (*Intestinal Obstruction by Meckel's diverticulum*), OESOPHAGUS (*Esophageal Pouches*), POST-MORTEM METHODS (*Body-Cavities, Abdomen, Diverticuli*).

**Divulsion.**—Rapid, forcible dilatation of a canal or hollow organ, such as the urethra or the cervix uteri.

**Dizziness.** See VERTIGO.

**Doble's Line.**—The dim line in the middle of the clear band of a muscular fibril. See PHYSIOLOGY, TISSUES (*Muscle, Structure of*).

**Dochmius Duodenalis.** See PARASITES (*Nematodes, Uncinaria Duodenalis, Ankylostomus Duodenale*).

**Docimasia.**—An examination or test (Gr. δοκιμάω, I prove), more especially of live-birth, the investigation of the heart and lungs, great vessels, and stomach, etc., in order to determine whether an infant has breathed after birth. See MEDICINE, FORENSIC (*Infanticide, Hydrostatic Test*).

**Dogs.** See ANTHRAX (*Lower Animals, Dogs and Cats*), DISTEMPER, RICKETS (*Morbid Anatomy, Puppies*).

**Dolichocephaly.**—The long-shaped skull, that in which the antero-posterior diameter is relatively long as compared with the transverse, with a cephalic index of less than 75. See ANTHROPOLOGY.

**Dolichocnemic.**—Long-legged (from Gr. δολιχός, long, and νῆμῃ, the part of the leg between the knee and the ankle), having the leg nearly as long as the thigh.

**Dolichohleric.**—Having the sacrum long in comparison to its breadth (from Gr. δολιχός, long, and ἑρῶς, the sacred bone).

**Dolichopellic.**—Having the antero-posterior (or conjugate) diameter of the pelvic basin as long as or longer than the transverse.

**Dolor.**—Pain or suffering. Various qualifying adjectives may be added, such as *dolor mitis*, slight pain, *dolor atrox*, agonising pain, *dolor capitis*, headache, *dolor dentium*, toothache, *dolor colicus*, colicky pain, etc. In the plural *dolores* signifies pains, and especially the pains of labour (*dolores ad partum*), which may be slight at first (*dolores præparantes, dolores præparantes*) and very severe in the later stage (*dolores conquiescentes*), and slight again after labour is over (*dolores puerperium or after-pains*). See LABOUR, STAGES AND DURATION.

**Domestic Measures.**—Since domestic measures vary so greatly in capacity, medicines ought to be dispensed as far as possible in bottles with graduated markings on the glass and the direction "one-sixth or one-twelfth three times a day," etc., but as a general rule the teaspoon holds about one fluid drachm, the dessert-spoon about two fluid drachms, the tablespoon about half a fluid ounce, the wine-glass about two fluid ounces, the teacup about five fluid ounces, the breakfast-cup about eight fluid ounces, and the tumbler about eleven fluid ounces. The drop cannot be safely regarded as exactly equivalent to one minim.

**Domicile, Law of.**—The place or country where a person's permanent home is, it may differ from his place of residence, for he may be living temporarily, but not indefinitely, in a foreign country, in cases of insanity, a lunatic "usually retains the domicile which he possessed at the time when he began to be legally treated as *non compos*."

**Donda Nduga.** See SKIN DISEASES OF THE TROPICS (*Tropical Phagelena*).—The name means literally "brother" or "companion ulcer."

**Donovan-Leishman Bodies.**—Small oval bodies (parasitic), probably protozoan in nature, found in the spleen, liver, blood, etc., in cases of malaria, chronic dysentery, kala-azar, low fever, etc.

**Donovan's Solution.**—Liquor arsenii et hydrargyri iodidi. See MERCURY

**Dorema Ammoniacum.** See AMMONIACUM

**Dormiol.**—A soporific or hypnotic medicine, said to be amylene chloral or dimethylethyl-carbamolchloral, it is a colourless liquid with an unpleasant taste, it is to be obtained in 50 per cent solution, and the dose is from 0.5 to 3 grams (8 to 45 grains)

**Dorsad.**—Towards the dorsal region

**Dorsal or Dorsalis.** See ANEURYSM (Lower Limb, Dorsal Artery), ARTERIES, LIGATURE OF (Dorsalis Pedis), BRACHIAL PLEXUS, SURGICAL AFFECTIONS OF (Dorsal Nerves), LABOUR, DIAGNOSIS AND MECHANISM (Transverse Lies), etc

**Dorso.**—In compound words *dorso-* signifies relating to the back, e.g. dorso-lumbar, etc. The expressions *dorso-anterior* and *dorso-posterior* refer to the relation of the back of the foetus to the uterus of the mother

**Dorsodynia.**—Muscular rheumatism affecting the upper part of the back

**Dosage.** See PRESCRIBING—A dose is the quantity of a medicine to be taken at one time, a maximum or full dose is the largest quantity which can be safely taken, while an infinitesimal dose is one so small as to be regarded as homeopathic. The study of doses is called *Dosology*

**Dotage.**—Senile feeble-mindedness

**Dothienenteritis.**—Typhoid fever (from the *dothi*, a small abscess, and *enteron*, a piece of gut or intestine)

**Double Consciousness.**—A morbid (somnambulistic) state in which there is apparently a double personality in the same individual, an extraordinary case is that of Miss Beauchamp, reported by Professor Prince of Boston (1906). See UNCONSCIOUSNESS (Double Consciousness), CRIMINAL RESPONSIBILITY, INSANITY, NATURE AND SYMPTOMS (Delusional, Alternative Personality)

**Double Monsters.** See TERATOLOGY, also LABOUR, FAULTS IN THE PASSENGER (Double Monsters)

**Double Vision.** See ALCOHOLISM (Sensory Phenomena), OCULAR MUSCLES, AFFECTIONS OF (Paralysis, Double Vision), TABES DORSALIS (Symptomatology, Ocular Paralysis)

**Doubt, insanity of.**—Doubting madness (*maladie du doute* or *monomanie raisonnante*) is that form of mental disorder in which the patient is morbidly scrupulous about the observance of minor details of accuracy in conduct

or abnormally timid in regard to the common risks of everyday life. See INSANITY, NATURE AND SYMPTOMS (*Insane Defects of Inhibition, Foule de doute* or *Sobering Insanity*)

**Douche.**—A jet or stream of water, simple or medicated, hot or cold, directed with some force against the surface of the body or into one of the canals opening on the surface of the body (e.g. the ear, nose, vagina, rectum, etc.), the name is also given to the instrument used, and to the act of applying the water or lotion. Air is occasionally used. See ABORTION (Treatment of Inevitable), BALNEOLOGY (Douche Baths, Hypothermal, Thermal, and Subthermal Douches, Scotch Douche, etc.), HYDROPATHY (Douches, Ascending, Descending, Spinal, Scottish, Air, Underwater), LABOUR, MANAGEMENT OF (Aseptic), LABOUR, INJURIES (Inversion of Uterus, Treatment), NOME, CHRONIC INFLAMMATION (Treatment, Nasal Douches), PUERPERIUM, PHYSIOLOGY (Management, Douching), PUPPARIUM, PATHOLOGY (Puerperal Infection, Prophylaxis and Treatment), UTERUS, INFLAMMATION OF (Chronic Metritis)

**Douglas, Mechanism of.**—A mode of spontaneous delivery, which occasionally happens in the case of transverse presentations of the child left to nature, described first by John C. Douglas (Dublin, 1819), "spontaneous evolution"

**Douglas, Pouch of.**—The pouch of peritoneum lying in front of the rectum (in the posterior part of the pelvis) and behind the bladder or the uterus (in the female subject), described by James Douglas (1675-1742), and named after him

**Dourine.**—A disease occurring in horses, transmitted by coitus (hence the synonymous term "mal de coit"), due to a trypanosome, and in some respects resembling syphilis (angioneurotic oedema, sclerosis of nervous system, spontaneous fractures, and dislocations). See PARAMITES (Protozoa, Trypanosomata, Trypanosoma of Dourine)

**Dover's Powder (Pulvis Ipecacuanhæ Compositus).**—Contains opium, ipecacuanha, and sulphate of potassium. See OPIUM, TOXICOLOGY (Opium and Morphine)

**"Dowsing" Method.** See HYDROPATHY (*Hot-Air Applications*)

**Dracontiasis.**—Guinea-worm disease, the disease due to the *Filaria* or *Dracunculus Medicinalis*. See FILARIASIS (*Filaria Medicinalis*)

**Dracontisomus.**—A variety of gastro-schisis, that teratological type in which there is median evagination of thorax and abdomen, twisting of the vertebral column, and a peculiar horizontal arrangement of the ribs (like the wings of a dragon or flying lizard)

**Dracunculus.** See FILARIASIS (*Filaria Medinensis*).

**Dragon's Blood.**—A resin (crimson in colour) found as an exudation from the fruit of the Rattan palm (*Calamus draco*), containing dracocalban ( $C_{20}H_{40}O_4$ ), and dracorenen ( $C_{20}H_{44}O_2$ ).

**Drainage.**—The removal of superfluous water and of sewage from launds and dwellings (see SEWAGE AND DRAINAGE), *surgical drainage* is the removal (by means of tubes or counter-openings or posture) of fluids from wounds or cavities (natural or artificial) of the body (see ASEPTIC TREATMENT OF WOUNDS, Drainage, BLADDER, INJURIES AND DISEASES, *Cystitis, Treatment, Drainage*, BLADDER, INJURIES AND DISEASES, *Tumours, Drainage*, MENINGITIS, TUBERCULOUS, *Drainage of Lateral Ventricles*).

**Drastics.**—Medicines acting quickly and violently, especially purgatives (*g.v.*), such as croton oil, jalap, elaterium, scammony, podophyllum, etc. See PHARMACOLOGY.

**Draught.** See HAUSTUS, PRESCRIBING.

**Dreams.** See SLEEP, NORMAL AND MORBID (*Hypnagogic State, Dreams*), HALL, MYOCARDIUM AND ENDOCARDIUM (*Symptomatology, Cerebral Symptoms*), MINN, EDUCATION OF, MORPHINOMANIA (*Effects, Dreams*).

## Dressings.

See also ASEPTIC TREATMENT OF WOUNDS, BANDAGES, FIRST AID.

THE question of the most suitable dressing for wounds sustained accidentally and intentionally inflicted received no final answer until a comparatively few years ago, when the principles and details of aseptic surgery were definitely elaborated. Many factors conspired to achieve this result, such as the invention of the microscope, and the discovery of the germ theory, with its widely ramifying and important results, but of all of them, the work of Lord Lister is paramount. His conception, founded upon a true scientific basis, and expanded on practical lines, which resulted in the employment of antiseptics, has been the means of advancing the rational treatment of wounds to its present and apparently final position, and the aseptic treatment of wounds has followed so rapidly upon the antiseptic treatment of wounds that there are to be found surgeons who treat their wounds upon the older lines, and text-books written but a few years ago by representative surgeons enunciate doctrines which to-day appear to be heretical to the modern aseptic surgeon.

In a practical work, a history of the endless variety of dressings which have been used from the earliest times is out of place, but those curious on this subject will find information in almost any surgical work published prior to the pre-Listerian era. Day by day the rivalry between different antiseptic dressings grows less

keen as the real value of the aseptic dressing is appreciated. Accordingly, the subject of surgical dressings becomes restricted to an account of the methods of obtaining a sterilised dressing, and a description of those comparatively few conditions in which an antiseptic dressing is more suitable.

With regard to the dressing of wounds which are expected to run an aseptic course and to heal by first intention, there is still some difference of opinion among surgeons. The difference lies in this, that one group of surgeons, feeling incredulous as to the possibility of having the skin aseptic in the neighbourhood of a wound, consider it necessary to apply to the wound an antiseptic dressing, one which, on account of its antiseptic properties, will prevent the development of pyogenic organisms in the vicinity of, or actually in the wound, while the other group of surgeons, relying upon their attempts to render the vicinity of the wound and the wound itself aseptic, content themselves with employing a dressing which has merely been sterilised. If this latter view is the correct one, then it of necessity follows that the subject of surgical dressings as applied to wounds believed to be aseptic and expected to heal by first intention becomes very much narrowed down. It therefore matters very little what is the nature of the dressing, so long as its chief function is fulfilled, viz that of shutting off the wound from the bacterial world until it is healed. As a matter of fact gauze is the material almost invariably selected, for, owing to its texture, it permits of easy sterilisation. It is comfortable, and readily absorbs any oozing from the wound but any linen or cotton fabric is almost as suitable.

Those surgeons, i.e. the large majority of surgeons, who advocate a simple sterilised dressing for a wound do so for the following reasons—

- 1 As both the wound and the dressing are aseptic, suppurative cannot occur in the wound unless pyogenic organisms are introduced from without, and a dressing of almost any material, if properly prepared and applied, will prevent this.

- 2 If pyogenic organisms have, during the operation, been introduced into the wound, an antiseptic dressing on the wound will have as little effect as an aseptic dressing in preventing their development.

- 3 The use of an antiseptic in the dressing is apt to be relied upon, and to diminish the stringency of the precautions which must be taken to secure true asepsis.

- 4 Its simplicity in preparation.

The preparation of the gauze, or other material, can be efficiently and rapidly carried out in hospital practice, more especially if the operating room is provided with a steam steriliser, in private practice aseptic gauze has

advantages over any antiseptic dressing, which at the time of use may actually be septic, and nothing is more readily obtained in even the smallest cottage than a pan of water in which the dressing may be boiled and sterilised in ten minutes, and also, from the fact that there is no antiseptic in the dressing which may be relied upon, it is probable that all the other precautions which are taken before an operation to secure asepsis will be more carefully and thoroughly carried out. It is well to apply to the wound several layers of gauze, covered by absorbent wool sufficient in quantity to enable equable pressure to be applied, to add to the comfort of the patient by preventing irritation or chafing from the bandage, and to further diminish the risk of pyogenic organisms from without coming in contact with the wound. And, after all, it will happen in only exceptional cases that infection of an accurately closed aseptic wound by pyogenic organisms from without will produce more than superficial suppuration in the wound.

In the event of asepsis not being maintained during the healing of the wound the typical symptoms will appear—pain and discomfort in the wound, and possibly a rise of temperature, which will be an indication that the wound requires to be dressed. If the septic change is due to a stitch abscess, removal of the offending stitch and the application of an antiseptic dressing may be sufficient to prevent further infection of the wound, this dressing should, so long as there is any trace of sepsis, be changed daily. For this purpose double evamde or carbolic gauze may be used, and some surgeons put a layer of aseptic gutta-serena tissue or jaconet over the wet gauze to further the absorption of pus from the wound. If the septic mischief is in the deeper parts of the wound several stitches should be removed, the septic part of the wound swabbed with iodoform gauze, and a similar dressing applied, so long as the wound remains septic, it should be swabbed daily with iodoform gauze, and a very successful method of preventing the discharge accumulating in the wound is to introduce into the wound a few strands of sterilised worsted which may be impregnated with aseptic iodoform. Mr Stiles of Edinburgh has done much to popularise the use of worsted in the treatment of wounds requiring drainage. It may be mentioned here that iodoform is not always aseptic, but its asepticity may be secured by keeping it in a 1 in 1000 solution of corrosive sublimate, no chemical change taking place, and the vessel containing the iodoform should, from time to time, be shaken up in order that the iodoform and the corrosive sublimate may be thoroughly mixed.

In wounds which are not expected to heal by first intention, *i.e.* those wounds in which a drainage-tube is necessary, those wounds in which, as in emergency operations, there is some

doubt as to their complete asepticity, those wounds, such as those left after castrating for lupus, in which the edges are not in contact, those wounds which cannot be with certainty rendered aseptic before or during the operation—to such wounds it is advisable to apply a dressing which contains an antiseptic in such quantity as to be effective without being irritating, and for this purpose the double cyanide or carbolic gauze may be used, or, better still, gauze which has been first sterilised and then wrung out of an antiseptic lotion, such as carbolic acid 1 in 40, lysol 1 in 100, or biniodide of mercury 1 in 1000. (It is almost superfluous to point out that an antiseptic dressing is deprived of its antiseptic value if it is impregnated with an antiseptic before sterilisation, for, during the process, the volatile antiseptic is driven off.) In such cases, it is well to change the dressing soon after the operation—on the following day at latest—for the antiseptic dressing becomes valueless as such so soon as the antiseptic volatilises. It is a mistake to apply large quantities of absorbent wool over a wound for the sake of soaking up the discharge from it, except in those cases, such as psoas abscess and empyema, in which the discharge is likely to be very profuse, and the practice of putting a quantity of absorbent wool as a pad on a dressing which shows signs of the discharge being “through” is to be deprecated, for, under such circumstances, the development and growth of pyogenic organisms is encouraged.

Summarising, then, it may be stated that in the case of wounds believed to be aseptic, in which the surrounding skin is believed to be aseptic, and which are meant to heal by first intention without the employment of a drainage-tube, and even, also, in the case of septic wounds which are believed to have been rendered aseptic during the operation, the best dressing consists of several layers of sterilised gauze, covered by absorbent wool and returned in position by a bandage. In wounds in regions in which it is no easy matter to render the skin aseptic, and where the dressings are apt to be soiled—such as the inguinal region—and in wounds not expected to heal without suppuration, it is advisable to apply a dressing of several layers of gauze which have been first sterilised and then dipped in one of the antiseptic lotions alluded to above. As manufacturers do not supply antiseptic dressings which have been previously sterilised except when specially ordered to do so, and even then, as their methods cannot be so reliable as those of a surgeon or of a trained assistant, it is to be recommended that the preparation of the gauze be carried out under the surgeon's immediate observation.

I am indebted to Messrs J F Macfarlan and Co of Edinburgh (in whose works Lord Lister spent much time when endeavouring to

produce a cheap and antiseptic dressing, and who were the first manufacturers to place such an article upon the market) for the following list of antiseptic dressings arranged in the order in which they are demanded —

<i>Gauzes</i>	Percentage of Antiseptic
1. Double Cyanide (Mercury and Zinc)	3
2. Carbolic	5
3. Iodoform	10-20
4. Sal Alembroth	1
5. Sublimat	2
6. Boric	20
7. Salicylic	4
<i>Absorbent Lints</i>	
1. Boric	45
2. Carbolic	5
3. Iodoform	10
4. Sublimat	$\frac{1}{2}$
5. Sal Alembroth	$\frac{2}{3}$
6. Double Cyanide	3
<i>Absorbent Wools</i>	
1. Sublimat	$\frac{1}{4}$
2. Sal Alembroth	$\frac{1}{2}$
3. Salicylic	1-10
4. Boric	.35
5. Carbolic	5
6. Double Cyanide	3
7. Iodoform	10

Though the same general principles underlie the application of dressings to any part of the body, whether for disease or injury, it occasionally happens that some modification is necessary, whether on account of the nature of the wound or the part in which it occurs.

1. Dressings for injuries to and after operations on the eye require, owing to the sensitiveness of that structure, to be of a non-irritating nature. Weak lotions are used here, and the antiseptic dressings employed are those in which the antiseptic is mild, such as boric lint or gauze, or those in which the percentage of antiseptic is low.

2. Dressings for the perineum and genital organs should contain an antiseptic, owing to the difficulty of securing asepsis of the parts, and owing to the risk of infection of the wounds by the feces or urine.

3. For artificial anus and fecal fistula carbolic tow is the dressing in general use, it is admirably absorbent, and the tar which it contains is most efficient in concealing or disguising the fecal odour.

4. Dressings for wounds from which the discharge is copious,—such as empyema and psoas abscess—should be abundant and antiseptic to arrest, as far as possible, the development of pyogenic and other organisms.

5. Dressings for syphilitic sores should, in

addition to the employment of an antiseptic dusting powder, such as iodoform or calamine, be impregnated with a mercurial antiseptic, and there is none better than corrosive sublimate. Black mercurial lotion is also used extensively for syphilitic sores.

6. Dressings for various diseases of the skin will be described in detail in the proper place. There can be little doubt that antiseptic dressings for those skin diseases in which pyogenic organisms are present have scarcely received that recognition from dermatologists which they deserve. It is unfortunate that the majority of chemists cannot be relied upon to dispense ointments the asepticity of which is undoubted; and, in those cases, *e.g.* the various forms of eczema, more rapid healing might often result if the ointment which might happen to contain no antiseptic were aseptic, but this can rarely be the case.

Certain untoward results occasionally follow the judicious employment of certain antiseptic dressings, or the employment of antiseptic dressings on certain individuals of peculiar idiosyncrasy. Similar results sometimes occur after the use of antiseptic lotions.

Boric acid may produce a localised erythema, in exceptional cases it has occasioned a widespread erythema. Its internal use is much more likely to cause toxic symptoms.

Carbolic acid should never be used in such concentration as to have a local irritating effect, but as a lotion, and in the older days when the carbolic spray was in everyday use, it not infrequently produced severe local and general symptoms, *e.g.* carboloria, which have proved fatal. The hands of some are very susceptible to carbolic acid as a lotion for instruments, rendering the skin hard and liable to crack, and therefore hard to render aseptic.

Salicylic acid, as an antiseptic dressing for wounds, is even more irritating than carbolic acid, and forms of eruption may be produced by its use, even in small percentages (erythematous, urticarial, vesicular, petechial, etc.).

Corrosive sublimate, when used in dressings, may cause much irritation of the skin, and it is not uncommon to find parts which have been prepared for operation by the application of a wet corrosive dressing covered with an erythematous eruption in which many minute pustules are present. Some hands are peculiarly susceptible to it, and its use by instrument-dressers not infrequently produces a painful condition of the matrix of the finger-nails. Corrosive sublimate cannot be too carefully used as a lotion for douching wounds or mucous surfaces, for, by its absorption, it readily produces toxic symptoms.

Iodoform may produce a rash of an erythematous, vesicular, or bullous character, and the idiosyncrasy of certain individuals is very marked in the use of this antiseptic. Wounds which

have been stuffed with an iodoform dressing, or cavities—such as tuberculous joints—which have been filled with an iodoform emulsion, occasionally result in the appearance of an erysipelatous rash, which rapidly disappears on the withdrawal of the antiseptic, but there are on record many cases which have resulted in death by the absorption of iodoform.

A not unimportant aspect of surgical dressing is that of rendering first aid to the wounded. There are now many thousand men and women in Great Britain who have received certificates of proficiency from the St John's or the St Andrew's Ambulance Association, and who consider themselves qualified (among other things) to apply a temporary dressing to those who have sustained wounds. The lecturers of these associations cannot be sufficiently impressed with the importance of teaching their pupils the value of interfering as little as possible with wounds, and instead of washing them out, even with an antiseptic lotion, of merely covering them with an aseptic dressing, however homely, which can be rapidly and easily obtained.

Von Bergmann found in the Russo-Turkish war that in 15 selected cases of compound comminuted fracture at the knee-joint, thorough disinfection of the surrounding skin, the application of a salicylic gauze dressing and a plaster of Paris splint proved sufficient for 14 to heal without suppuration.

For the temporary treatment of wounds on the battlefield, it can scarcely be expected that an aseptic dressing will be at hand, and army surgeons have usually to employ an antiseptic dressing, but it is desirable that those dressings should be first sterilised.

**Drinking-Cup.** See INVALID FEEDING (*General Serving of Food*)

**Drinking-Water.** See CONSUMPTION (*Causes, Drinking-Water*), THYROID GLAND, MEDICAL (*Goitre, Etiology*), TOXICOLOGY (*Plumbism, Drinking-Water*), TYPHOID FEVER (*Etiology, Water Supply*)

**Droitwich.** See BALNEOLOGY (*Great Britain, Mineral Waters*), MINERAL WATERS (*Mineral Saline Waters*)

**Dromotherapy.**—Treatment by running exercises in the attitude of flexion ("In course on flexion"), the arms are flexed at the elbows and held well back, the head is slightly elevated, and the spine kept straight, it is recommended in cases of disease due to slowing of nutrition and the troubles arising therefrom, and it forms a useful variety of respiratory gymnastics in chronic disease of the lungs (F. Regnault).

**Dromotropic.** See BATHOTROPIC INFLUENCE

**Drop.** See DOMESTIC MEASURES (*Minim*)

**Dropsy.** See also ABDOMINAL ANEURYSM (*Pressure-Symptoms*), ASCITES, BERIBERI, DIABETES MELLITUS, DROPSY, EPIDEMIC, HEART, MYOCARDIUM AND ENDOCARDIUM (*Effects of Cardiac Disease, Dropsy*), HEART, MYOCARDIUM AND ENDOCARDIUM (*Symptomatology, Dropsy*), LARDACIOUS DEGENERATION (*Effects*), LUNG, TUBERCULOSIS OF (*Complications, Integumentary System, Edema*), NEPHRITIS (*Clinical Features, Acute, Chronic*), OVARIES, DISEASES OF (*Symptoms of Ovarian Tumour*), PREGNANCY (*Affections of the Amnion, Hydramnios*), SCARLET FEVER (*Complications, Diseases of the Urinary System*)—Dropsy is defined as the accumulation of fluid in the subcutaneous tissue and serous cavities of the body.

Various terms are applied to this condition as met with in different situations, e.g. ascites, hydrothorax, hydrocele, and hydrocephalus. The terms oedema and anasarca are also employed, the former indicating local dropsy in subcutaneous tissue, the latter, widespread general dropsy.

Dropsy is a symptom, and is usually associated either with primary cardio-vascular or renal disease. In both instances the diagnosis of the case can readily be arrived at. Although occasionally there are mixed cases, where the cardio-vascular lesions are secondary to renal disease, cases are occasionally seen with well-marked dropsy of the lower extremities, apparently of causeless origin. One such case, a girl of sixteen, has come under the writer's observation. Here cardiac and renal diseases could be excluded. Similarly filariasis, and a careful pelvic and other examination revealed nothing that might by pressure or otherwise produce the disease. The treatment of this case was as unsatisfactory as its nature was doubtful. The significance and treatment of dropsy will be considered in the articles dealing with the "Heart" and "Nephritis," and the local dropsies under their respective headings.

The only outstanding local dropsy meriting special attention is ascites, and this is fully considered in vol. i p. 285.

The object of the present article is to give a general outline of the etiological factors at work in the production of dropsy.

Although much could be written on this subject, we know comparatively little of definite practical importance. Every clinician is familiar with cases of advanced cardio-vascular disease without dropsy, and of other cases less serious in nature where oedema may be a prominent feature. The same can be said of various forms of kidney disease, and so far, there is no adequate explanation forthcoming for these anomalies. The question really resolves itself into the fundamental one of the primary factors concerned in the movement of lymph, and then

to a consideration of the influence of various diseased states on these different factors. On neither of these points can final and complete statements be made. The physical and chemical natures of the fluids are discussed elsewhere (See "Fluids, Examination of Pathological").

The agencies at work in promoting the movement of lymph will be considered under "Lymphatic System," and it will suffice to indicate here the directions in which these agencies may be modified.

For convenience these will be stated categorically, but in nature no such arbitrary line can be drawn, as not one but many factors are frequently at work in any given case.

I. Cardio-vascular Disease.—Any condition which induces either increased forward (arterial) pressure, or an increased backward (venous) pressure, especially the latter, predisposes to the occurrence of dropsy. In both cases the primary cause is probably a vital alteration in the capillary endothelium, and in both, physical causes the results of pressure are also at work. Experiments have shown that obstruction to the venous return in a limb even when complete is not sufficient to induce dropsy, as the lymphatic system possesses a remarkable compensatory power.

If the increased venous pressure is associated with an increased afflux of arterial blood, such as might arise from vaso-motor disturbance, the tendency to dropsy is much increased.

In all such cases the influence of any associated altered quality of the blood, whether due to a simple hydiæmia, presence of salts in excess, rendering diffusion more easy, or the presence of various toxic bodies, has to be carefully considered.

A watery state of the blood alone has been shown by experiment to be insufficient to produce dropsy until the hydramic condition has lasted a sufficient time to alter the vitality of the endothelium. Various other experiments have been made by physiologists on the effects of the injection of glucose and other bodies into the blood-stream, and the results go far to indicate the important part played by altered states of the blood.

II. Lymphatic System.—With regard to the lymphatic system experimental observations have shown that obstruction to a lymphatic trunk is not in itself sufficient to induce dropsy if the other parts of the vascular system are intact.

III. Nervous System.—The influence of the nervous system is undoubted, but the exact mode of action is uncertain. We know that there are at least two directions in which the nervous system may act: (a) Through alteration of the vaso-motor mechanism involved, (b) in virtue of the trophic influence on the tissues. Of these two the latter, although more indeterminate, is probably the more important, and it is to differences in the vital absorbent power

of the tissues that we must mainly look in explanation of the occurrence of dropsy in many cases.

Local dropsies have, as a rule, local causes, but apart from the influence of gravity and the presence of a definable obstruction of a vessel, as by a thrombus, in every case it is advisable to think of, *seriatim*, the nature of the arterial afflux (of the local pulse), the freedom of the venous return, the absorbent power of the tissues, especially the endothelium and the general neuro-muscular tone of the individual.

*Diagnosis*.—While the recognition of dropsy is easy, great difficulty is frequently experienced in apportioning due significance to the important causal agents in any given case. It is important to recognise this with a view to accurate prognosis and successful treatment. Great regard should be paid to the state of general blood-pressure, and the working power of the right and left heart should be separately investigated. The existence of toxic bodies in the blood may frequently be inferred from the quantity and quality of the urine voided, corroborative evidence being obtained from a study of the case as a whole, and especially the state of the central nervous system.

*Prognosis*.—This depends entirely upon the cause and upon amenability to treatment. A consideration of the various points referred to under diagnosis are of first importance.

*Treatment*.—The first indication is to test the cause, and if dependent on any specific toxic body, *e.g.* beriberi, the treatment appropriate to that disease must be adopted. Otherwise the treatment must be conducted on the general lines indicated by the diagnosis.

Complete rest in bed, judicious use of bandages, and the use of various cardiac tonics, and careful attention to the functions of the skin, bowels, and kidneys, are the remedies of greatest service. An occasional saline is the best form of purgative. It is also advisable to limit the amount of fluid ingested, and in severe cases benefit is obtained by aspiration (*q.v.*) or by the use of Southey's tubes, care being taken to use these remedies under very careful aseptic conditions, other appropriate general or nervous tonics are called for.

## Dropsy, Epidemic.

See also BERTIERI

BETWEEN the years 1877 and 1880 there broke out in Calcutta a peculiar disease to which the term epidemic dropsy was applied. The disease only occurred during the cold weather, and a similar outbreak was noted in Mauritius and Assam in 1878, and at Banca and South Sylhet in the cold season of 1878-79. The best account of the condition has been given by McLeod, who described the cases as seen in Calcutta (*Trans. Epid. Soc.* vol. xii).

The mortality there was as high as 20 to 40 per cent, while in the other districts the



mortality was trivial in comparison with these figures.

At the time of its occurrence the condition was regarded by many to be merely that type of beriberi in which dropsy is a pronounced symptom, the nervous phenomena being very slightly marked, but McLeod concluded that it was a distinct entity. As no further outbreaks have been recorded, our knowledge of the disease has not increased. Little is known regarding the morbid anatomy and etiology, but the fact of its communicability and the histories of the successive outbreaks furnish evidence in favor of a germ theory.

The disease ran a course of from three to six weeks. Dropsy, anemia, and fever formed the cardinal symptoms, these usually being accompanied by great general weakness, wasting, breathlessness, diarrhoea, vomiting, and various nervous symptoms. The amount of anemia and dropsy present gave rise to the term *acute anæmic dropsy*. The dropsy was usually, but not invariably, preceded by the febrile and other symptoms hereafter referred to. The onset of the oedema was sudden, and first showed itself in the legs, and in severe cases soon became general, with the development of extensive pleural and pericardial effusion with oedema of the lungs. A distinct tendency to recurrence of the dropsy after its disappearance was noted in some cases. A remittent type of fever was present, not usually high, but varying from 99½° F. to 102° F., and the time of its occurrence bore no apparent constant relationship to the development of the dropsy. Alimentary disturbances, notably diarrhoea and vomiting, and also nervous symptoms, *e.g.* anesthesia of the skin, itching, and paresis of muscles, were occasionally met with, but were not common. An erythematous skin eruption not infrequently appeared about a week after the oedema. In fatal cases death was sudden, and was due to the pulmonary and cardiac complications.

The points on which stress was laid in support of the view that the disease is distinct from beriberi are—The acuteness of the affection, the presence of well-marked fever, and the skin eruption, but it must be admitted that further information is wanting before the distinction can be fully and finally drawn (*vide* "Beriberi").

**Treatment.**—From our ignorance of the cause, treatment must be conducted simply on the general medical principles laid down in that article.

**Drowning.** See ASPHYXIA, MEDICINE, FORENSIC (*Death from Asphyxia*), MEMORY IN HEALTH AND DISEASE (*Hypemnesia*)

**Drowsiness.** See BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Anæmia*), BRAIN, SURGERY OF (*Cerebral Abscess, Drowsiness*), STOMACH AND DUODENUM, DISEASES OF (*General Symptomatology, Remote Symptoms*)

## Drug Eruptions.

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See also BELLADONNA, BROMISM, DERMATITIS TRAUMATICA ET VENENOSA, PHARMACOLOGY, PURPURA (*Toxic*), RUBELLA (*Drug Rash*), TOXICOLOGY, and under some of the various Drugs themselves

THAT eruptions follow the external or the internal use of drugs in certain classes of individuals is a fact with which all dermatologists are familiar, but in the intensity of the reaction produced variations occur within wide limits, so that, in conjunction with the personal idiosyncrasy, frequent modifications of type may be expected, and, indeed, are almost invariably found. In certain persons the exhibition of a given drug in any form acts as a poison, while in others, and probably the majority, no toxic effects are produced, and it must be borne in mind in this connection that the occurrence of certain constitutional diseases modifies in a marked degree in many cases the action which occurs during administration, or even produces a sort of immunity—in other words, creates in the individual a certain tolerance which could not be acquired under the normal conditions of freedom from disease. This is best exemplified in the increased resisting power to the action of both mercury and the salts of iodine shown by the vast majority of syphilitic patients. It is obvious, therefore, that in considering the general question of drug eruptions, two most important factors must be constantly borne in mind—first, the natural, or acquired, immunity enjoyed by certain individuals, and, secondly, the peculiar susceptibility from which other persons suffer—the cause in either case being equally undetermined and undeterminable.

Drug eruptions are divided by natural lines into two great classes (a) *traumatic dermatoses*, those produced by the action of irritant or poisonous substances coming in direct contact with the skin, which may be parasitic, animal, vegetable, or inorganic in nature, and (b) the *toxic dermatoses*, those cutaneous manifestations which follow in certain individuals the internal administration of various drugs. The former class will only be incidentally alluded to here, as it has been already dealt with (*see* "Dermatitis

Traumatica"); and our attention may therefore be wholly fixed (A) On the nature and varieties of the eruptions produced by the ingestion of medicinal substances, and (B) in detail, on the various more important drugs associated with the occurrence of skin lesions. It is clear that in an article of this scope no exhaustive treatment of the subject is possible, and therefore only the more frequent forms of drug eruption will be dealt with.

A. Considering the variety of substances which in their assimilation or elimination are capable of giving rise to such eruptions, it is not a matter of wonder that the forms assumed do not conform to any one type, but are in the widest degree polymorphous and interchangeable. Thus similar lesions may be produced by different drugs in different individuals, or eruptions widely different produced by the same drug in persons whose individual susceptibility defies computation. In fact, in these cases no class of skin lesion has been left unexampled, and, while in the majority of cases they are erythematous in type, they are capable of all the variations which are met with in that large and inconstant group. They may simulate the acute exanthems of measles, variola, scarlatina, or rotheln; they may mimic with startling accuracy the forms of cutaneous erysipelas; they may be erythematous, papular, urticarial, vesicular, pustular, bullous, or even gangrenous, according to the degree of reactionary inflammation elicited in the given individual. Hence, in many instances, the difficulties which surround the diagnosis and the errors into which those unacquainted with the polymorphous lesions have been led. Thus cases have been recorded in which fatal results followed the administration of the iodides, the patients succumbing to a bullous and gangrenous dermatitis induced by progressively increasing doses administered for the cure of an imaginary syphilide.

The following tabular list, in which the effects following the external application of drugs have been compared with those resulting from their internal administration, may prove useful for purposes of reference—

#### TYPES OF ERUPTION AND DRUGS ASSOCIATED WITH THEIR OCCURRENCE

##### 1 Erythematous

- (a) *From External use*—Aconite, Arnica, Balsam of Peru, Boric Acid, Carbolic Acid, Chrysarobin, Croton Oil, Iodoform, Oil of Cade, Tar, Turpentine.
- (b) *From Internal use*—Antipyrin, Antitoxin, Arnica, Arsenic, Belladonna, Boric Acid, Bromides, Chloral Hydrate, Copaiba, Cubebs, Hyoscyamin, Iodides, Morphin, Quinine, Phenacetin, Rhubarb, Salicylic Acid, Stramonium, Sulphonal, Tar, Turpentine.

2 Papular, Vesicular, Pustular (*mixed, confluent, and exaggerated forms*).

- (a) *From External use*—Aconite, Antimony, Arnica, Bichromate of Potash, Cantharides, Carbolic Acid, Chrysarobin, Oil of Cade, Croton Oil, Iodoform, Ipecacuanha, Mercurial Salts, Rhus Toxicodendron ("poisoning"), Sulphur, Tar, Terebene, Thapsia.
- (b) *From Internal use*—Aconite, Antimony, Arsenic, Borax, Bromides, Cannabis Indica, Chloral Hydrate, Copaiba, Cubebs, Digitalis, Ergot, Iodides, Iron (principally the iodide), Quinine, Sulphur, Terebene.

##### 3 Urticarial.

- (a) *From External use*—Aconite, Balsam of Peru.
- (b) *From Internal use*—Antipyrin, Antimony, Bromides, Copaiba, Iodides, Quinine, Morphium, Santonin, Sulphonal.

##### 4 Pigmentary

- (a) *From External use*—Chrysarobin, Picric Acid.
- (b) *From Internal use*—Argenta Nitras, Arsenic, Antifebrin.

Regarding the *etiology* of these eruptions but little is definitely known. It has been held that they are due to an elimination of the toxic substances themselves, or of some subtle compounds formed from them in the tissues, by means of the cutaneous glands, especially the sebaceous follicles, and support of this view has been sought for in the fact that the tendency towards the development of such lesions is largely aggravated in cases of renal or cardiac inadequacy. This, which means a compensatory eliminative activity of the skin glands, presupposes a certain degree of saturation of the system with the drug, but clinical experience goes to prove that toxic dermatoses are just as likely to occur in cases where only a few, and those minimal, doses have been given, as in cases where a certain amount of accumulation has taken place from prolonged and continuous administration. Stress has also been laid on the fact that in some cases traces of the offending drug (e.g. biomine, iodine, etc.) have been detected in the secretions of the skin, but these observations have by no means been universally confirmed, which would be essential to the establishment of the postulate; while in nearly all cases evidence of the elimination of the drug in the urine can be obtained. Further, histological examination has shown conclusively that the cutaneous lesions are not confined to, or even mainly manifested in connection with, the glandular apparatus of the skin. The neighbourhood of the blood-vessels shows the most pronounced changes—general hyperemia, and small multiple inflammatory areas, which often become abscesses of microscopic size, while the glandular structures—the

oil glands, and more especially the sebaceous glands—are only to a small extent, and that secondarily, affected. It may also be pointed out that the distribution of such rashes is often, indeed in the majority of cases, confined to a limited area, and does not present the universality of a circulatory poison, and Morrow has drawn attention to the fact that very many of the drugs associated with skin eruptions have a known influence upon the nervous system, either by direct action upon the peripheral nerve endings or on the neuro-vascular centres. We are therefore driven by a process of exclusive reasoning to the conclusion that in all these exanthematous rashes produced by drugs we have to deal with “a vaso-motor neurosis either from reflex irritation (i.e. from the peripheral nerve endings) or from direct action on the vaso-motor centres” (Crocker). But above all it must be constantly borne in mind that, in all cases almost, the determining factor is the “personal equation,” than which no more variable or elusive quantity can be found in studying the causation and remoter consequences of disease.

A word must be said here as to the *differential diagnosis* of drug eruptions in general from the particular exanthematous rashes they simulate, —a point of the greatest clinical importance, and, indeed, the only one which raises these manifestations above the level of pharmaceutical curiosities. In the former case there is a history of the administration of the drug, and there are, as a rule, no constitutional complications. In the case of copious rash, which most closely resembles that of measles, the eruption supervenes suddenly without any precedent fever or coryza, though it should be noted that flushing of the face and injection of the conjunctiva may occur. In the scarlatiniform rash produced by belladonna, quinine, etc., there is no fever, no sore throat, though there may be vivid redness of the fauces, and no glandular complication, and the same remark applies equally to cases where the eruption more closely resembles that of German measles. It is hardly possible that any of the pustular forms could be mistaken for variola, but the surest way of escaping mistakes in all cases is an acquaintance with the perfect mimicry with which the exanthematous rashes are copied in many of the forms of eruptions due to drugs. The avoidance of error is only possible where the knowledge of its likelihood exists.

#### B DRUGS WHICH PRODUCE TOXIC ERUPTIONS

—In dealing with the drugs which are most frequently concerned in the production of these varying eruptions in fuller detail, we have placed them for convenience of reference in alphabetical sequence. The symptoms associated with the development of each will there be given, and also the indications for any special, general, or local treatment, which has either proved of specific value or of benefit in alleviating symp-

toms, under the heading of each particular drug. The general principles of treatment will be referred to at the end of this section.

*Antifebrin* (Acetanilide).—A rash, somewhat resembling that of measles, has been described as following after the internal administration of this drug, even in doses of ten grams. When its use has been continued over a lengthened period, or given in larger doses over a restricted time, a peculiar cyanosis is induced which has been described as resembling a “slaty-coloured anemia.” There are no constitutional symptoms, and no alteration in the colour of the urine.<sup>1</sup>

*Antipyrin* (phenazone), when given internally, produces in a large proportion of cases a cutaneous eruption—the proportion varying from 2 per cent in men to 7 per cent in women. It is most frequently morbilliform in type, affecting the extremities more than the flexor aspects, and the extremities more aggressively than the body. Its symmetry is usually well defined, and it is associated with profuse sweating, moderate itching, and a certain amount of subsequent desquamation. A distinctly urticarial type has also been observed, and in exceptional cases the form assumed has been that of a purpuric or bullous eruption. These various results are probably brought about by the action of the drug in causing paralysis of the vaso-motor nerves, and thus leading to the presence of a largely increased amount of blood in the vessels of the skin. The eruption generally disappears rapidly on the immediate discontinuance of the drug, more slowly when it is given in gradually decreasing doses.

*Argenti nitras*. The peculiar bluish-grey discoloration of the skin produced by the long-continued administration of nitrate of silver, and embraced in the term “argyria,” is the most commonly observed result of the drug, though Charcot has described an itching and erythematous-papular eruption as having followed its use in a few cases. It must be borne in mind, however, that its internal use as a medicine is not necessary for the production of the skin discoloration, as this result may follow the habitual application of a solution of the salt to mucous surfaces, as, for example, in chronic throat affections. The deposit of silver, or silver combination, in the skin “is always confined within exact limits to the margin of the connective tissue, and specially affects the elastic fibres and the resisting limiting membranes, avoiding altogether the protoplasmic parts of the connective tissue, and the epithelium with its appendages” (Umma). It is stated that the prolonged administration of iodides given in very dilute combination with mineral waters (Vals, Vichy, etc.), along with massage in warm baths, aids in the elimination of the silver, but this is

<sup>1</sup> The drugs which induce marked alterations in the urine will be referred to under “Urine.”

doubtful, and when the deposit has attained a certain limit, its removal is hopeless.

*Arsenic* produces skin manifestations of varying degree either by local contact or by ingestion. The former are especially seen amongst those who work in it, either in the crude form, or in some of those numerous articles of commerce in the manufacture of which it plays so important a part. Thus they are met with in those engaged in the manufacture of artificial flowers, cart-board boxes, etc., and in those who wear stockings or other articles of underclothing dyed with the cheaper colouring matters which often contain arsenic in a poisonous degree. In other cases these eruptions have followed the use of skin lotions and cosmetics. The results of its internal administration are various, and numerous types have been described. An *erythematous* type resembling erysipelas, and often becoming vesicular, affecting mainly the face and eyelids, which become congested and oedematous, is one of those most frequently met with. Another—the *papular* form—appears mainly on the face, neck, hands, and about the genital organs. At first the papules are few in number and small, occurring in scattered groups which subsequently either enlarge or coalesce to form disseminated patches of irregular outline that may bear a close resemblance to a papular syphilide, though the coppery hue is less pronounced or absent altogether (Imbert-Jourbeire). The *urticarial* type has been frequently observed, and differs in no essential particular from the well-known wheals and associated symptoms of ordinary nettle-rash. The *vesicular* form is now well recognised, sometimes resembling eczema of an obstinate character, and such as one meets with in ordinary cases of “weeping eczema.” Hutchinson first directed attention to the comparatively frequent occurrence of attacks of herpes zoster (“shingles”) in persons taking arsenic. His observations have been extended and confirmed by Nielsen, who found that of 557 cases of psoriasis treated with arsenic in the Copenhagen General Hospital, nearly 2 per cent (180) suffered from herpes, whilst among those treated without arsenic not a single case occurred. The relation of arsenic to the production of peripheral neuritis is well known, and furnishes an explanation of the etiology of zoster in these cases. A *pustular* form has also been observed to follow the administration of the drug, finding its termination in the development of crusts or ulcers which heal slowly with permanent scarring. Morris has noted the occurrence of boils and carbuncles, an observation confirmed by others, which points to a graver degree of pyrogenic infection, or may be due to an accidental inoculation. Thickening of the skin of the palms and soles is also seen, the induration beginning around the sweat follicles, and gradually extending thence peripherally until

a uniform condition of *keratosis* is produced. Hutchinson has directed attention to the tendency this induration shows to ulceration and to the subsequent ultimate development of epithelioma. It is obvious, therefore, that in the multiplicity of lesions produced by arsenic great discrimination is essential, the ultimate test in every case being the disappearance of the eruption on withholding the drug. In the pustular or ulcerative condition the use of soothing or antiseptic lotions may become a necessary adjunct to treatment.

*Belladonna* (atropin) produces a diffuse erythematous blush which is mainly limited to the face, neck, and upper part of the thorax, and which only rarely becomes generally distributed. It is of a vivid redness and closely simulates the exanthem of scarlatina. It occurs for the most part (leaving aside cases of poisoning) in young children with clear, transparent skins, is extremely evanescent in duration, and is *not* followed by desquamation. Crocker states that he has rarely seen the eruption following medicinal doses, but agrees with its occurrence in cases of poisoning by the drug. The use of atropin in eye practice is occasionally followed by a severe inflammation which strikingly resembles erysipelas. Fox has called attention to the fact that the diffused scarlatiniform rash may be seen occasionally to follow the external application of belladonna in some of its preparations, such as the emplastum or glycerin. The point of chief clinical importance is to discriminate between this drug rash and that of scarlatina, but the factors already mentioned, when no epidemic is prevalent, especially the absence of prodromal symptoms, the fleeting nature of the erythema, and the usually almost normal temperature, can hardly, if carefully weighed, leave any possibility of error.

*Benzoin*.—In the form of Friar's balsam, ordered for inhalation, Fox has observed the occurrence of a purpuric erythema, which was confluent upon the trunk, but more discrete upon the extremities. Benzoic acid given in small quantities has been known to produce a rose-coloured maculo-papular eruption, which disappeared on discontinuance of the drug, and several cases have been noticed where erythematous rashes, attended with itching and followed by slight desquamation, have resulted from the administration of ordinary medicinal doses of benzoate of sodium.

*Boric Acid*.—Erythematous rashes, followed in a certain number of cases by even fatal results, have been reported as a consequence of the absorption of boric acid from washing out cavities, generally serous, with large quantities of the drug in solution. Impetiginous eruptions have also followed its internal administration. The borate of sodium, given in 5-grain doses over a long period, in cases of epilepsy, has been observed by Gowers and Laving to give rise to

a psoriasis which differs in no respect from the usual chronic type

**Bromine and the Bromides**—Probably no variety of drug eruption is more frequently observed, or is capable of wider variation, than that due to the administration of the salts of bromine. In general characteristics the rashes produced resemble closely the group of iodine eruptions to be considered subsequently, but they always present shades of difference to the accustomed eye, and two important points of differentiation in general are these—they are always much slower in development, and they are much less likely to become confluent over a large area and to assume a bullous or gangrenous type. In form they range over the whole nomenclature of primary skin lesions, and may be pustular, erythematous, urticarial, bullous, or squamous in type—the above sequence indicating approximately the relative order of frequency.

The *pyridio-pustular* form—known as “bromic acne”—is the most frequent, and occurs in about 75 per cent of all persons taking potassium bromide. It appears most commonly on the face, chest, back, and in the neighbourhood of the hair follicles of the thigh and leg, and it may vary between the extremes of being in the simplest form *discrete* and *acne-like*, and in the gravest form *confluent* and *fimular*, though between these extremes many degrees of variation will be found. The pustules are yellowish in colour, seated on an inflamed base, and differ from those of simple acne in that they appear without the necessary pre-existence of comedones, and they are not confined in distribution to regions where glandular structures naturally occur. They vary in size within wide limits, and in the confluent type they merge into each other, forming conglomerate groups, and gradually increasing in size by the extension of their margins until they may eventually cover a considerable area. In the meantime the contents dry up and become covered with brownish crusts which are elevated on a zone of surrounding erythema, and which tend to become associated with a distinct and definite hypertrophy of the underlying papillary bodies. This is the form to which Unna has specifically applied the term *bronchodermatopustulosis*. The discrete form differs from ordinary acne in that the inflammation is generally more acute, the lesions suppurate more rapidly, and the contents are of a more sero-purulent type. In the confluent form the presence of numerous pustular points on a raised, soft, dusky-red, and painless base, and the disappearance of the lesions under a scab without the formation of a scar, but with a slightly persisting reddish-brown stain, serve to differentiate it from all other eruptions except those due to the iodides (Crocker).

It is probable that individual idiosyncrasy plays a less important part in this than in the

majority of other drug eruptions, hence its universality and the uniformity of its main characteristics. It is known that elimination of bromine takes place through many glandular tissues, principally the kidneys, and in a less degree by means of the salivary, sweat, and mammary glands, when the latter are functionally active, as witness the many recorded cases in which symptoms of bromic eruption have occurred in infants suckled by those taking the drug. On this analogy it has been argued that the skin manifestations are due to its elimination by the sebaceous glands, but many facts previously referred to throw doubt on the genuineness of this hypothesis. In connection with this theory attention may be directed to the fact, pointed out by Crocker, that the eruption very frequently begins in scar tissue, being even occasionally limited to a favourite site being in vaccination scars. It is needless to point out how entirely this is opposed to the fundamental premises on which the theory of glandular elimination is based. It may be mentioned here, in order to avoid error, that in some cases the rash only makes its appearance after the discontinuance of the drug, and that also, as in the case of the iodine eruptions, where there is idiosyncrasy, small doses are more likely to evoke the symptoms than larger ones.

In this, as in most of these drug affections, the main indication of treatment is to stop the cause. The administration of liquor arsenicalis, 3-5m, two or three times daily, is said to act as an eliminant. Local applications, such as lead or evaporating lotion, or salicylic acid (2i j ad 5j) on lint covered with gutta-serena tissue may be required when the inflammation is severe or the skin surface broken. Liquor arsenicalis, or some preparation of ammonia, such as the carbonate or the aromatic spirit, added to the prescription containing the bromide, is said to markedly diminish if not entirely prevent the tendency towards the development of the eruption. In regard to the stoppage of the bromide in these cases it should be remembered that eminent authorities have laid down as a positive law that a direct relation exists between the intensity of the skin eruption and the efficacy of the drug as a remedy in the disease for which it is administered. This again is a point which can only be determined by careful and individual observation.

**Cannabis Indica**—In one recorded case an acute vesicular eruption of very wide distribution followed a dose of one gram of the extract. It subsided in the course of a few days, the vesicles drying up and leaving crusts which gradually fell off (Hyde). Edema of the face has been noticed as one of the symptoms of chronic intoxication with this drug.

**Chloral Hydrate**—The local effect of chloral as a cutaneous vesicant is well known, and it has been largely recommended in certain cases

as a substitute for the preparations of cantharides, but considering the large number of affections in which it is employed internally it cannot be generally recognised as a frequent producer of cutaneous eruptions. In its special features the rash usually appears within a short period, has only a very limited duration, and is unaccompanied as a rule by constitutional or local disturbances, though in some cases itching or irritation has been observed. The rash when it occurs is generally erythematous in type, but may present the appearance of dusky-red papules surrounded by an area of diffused redness extending about the affected part, which are mainly the face, neck, and neighbourhood of the large articulations. In some cases it may be morbilliform in character, and urticarial, vesicular, and petechial forms have also been described. A remarkable relationship exists in all the forms between the ingestion of food, and more especially of alcohol, and the development of the rash. This has even been observed in children taking the drug, when alcohol, in the form of a tincture, has been prescribed in combination. In all cases the effects produced are probably due to the influence which chloral exerts upon the vaso-motor system.

*Copaiba*.—The type of the eruption is mainly erythematous and papular, or more essentially morbilliform, with a special predilection for the neighbourhood of the hands, arms, feet, knees, and abdomen. In its most characteristic form it consists of "rose-coloured, irregular patches, grouped or discrete, and only just perceptibly raised above the surface." The redness disappears completely on pressure. Intense itching may be present, and a form which successfully simulates urticaria is well known. The rash fades rapidly on withdrawal of the drug, and is succeeded by slight desquamation. These forms are commonly met with, and may follow the administration of the drug for genito-urinary conditions or for chronic chest affections. It is probable that in the majority of cases the cutaneous manifestations are due to the elimination of the volatile oil by the sweat glands, but in the urticarial form they may be due to the gastric irritation which is so constantly associated with the use of the drug, and which is so largely a factor in the production of ordinary urticaria. As curiosities of dermatology, vesicular, bullous, and petechial eruptions have been described as sequelæ to the administration of copaiba. Clinically its importance consists in the differential diagnosis between the drug eruption and the rash of measles, of rubella (rubella), or of an erythematous syphilide.

*Cubeba*, which is much less extensively used, in this country at least, than copaiba, produces in a few cases similar cutaneous manifestations which do not require separate description.

*Ergot*.—The subcutaneous injection of ergot

not infrequently induces a painful, dark-coloured swelling at the point of puncture. Erythema, with swelling of the parts, vesicular and pustular eruptions, and gangrene of the extremities, may occur from its internal administrations, but the latter rarely from its use in medicinal doses.

*Iodine and the Iodides*.—Characteristic types of these eruptions, to be described presently in detail, have resulted from the external application of iodine, such as repeated painting of a part with the tincture or liniment, or the injection of some such preparation to excite inflammation in a serous cyst, but they are most frequently observed to follow the ingestion of some of its alkaline salts, even in the most minute doses—and iodide of potassium, the most frequently administered, may be taken in its effects for the whole group. It may be here incidentally stated that, according to many observers, the sodium salt is the least and the ammonium salt the most active in producing cutaneous manifestations, and that in many cases of pronounced idiosyncrasy the strontium salt may be taken with impunity. In all the varieties of drug eruptions no more conglomerate group can be found, and it is just this absence of clinical unanimity which renders their recognition difficult and at the same time imperative, for in several cases fatal results have followed from ignorance of the cause which determined the skin lesions. As Morrow well puts it, "iodide of potassium may be continued, possibly in increasing doses, for the very condition which it has caused."

No more polymorphous eruptions are known to dermatology than those due to the iodine compounds. As in the case of the bromine salts, the commonest form is the pustular, but other types met with are the erythematous, vesicular or bullous, urticarial, and purpuric varieties, the two latter belonging to the category of dermatological curiosities.

The most frequent of all is that to which Unna has applied the term *iododerma pustulotuberosum*. The rash begins as small papules rapidly becoming pustular, and occurring most frequently on the face, upper part of thorax in front and behind, and sometimes on the extremities. In its early stages it closely simulates the bromide eruption, to which the false term "bromic acne" has been commonly applied. These lesions may remain discrete or become confluent, and so merge into some of the more aggravated types. That the eruption is not due to glandular elimination has already been stated, its distribution does not always correspond to that of the supposed eliminating glands, nor does it present the universality and symmetry necessary to a circulatory poison. Microscopic examination of excised portions of skin has further shown that far from elimination taking place from the sebaceous follicles, as would be evidenced by marked changes there,

ie foci of greatest inflammation are situated somewhat superficially in the skin, where they aggregate into microscopic abscesses. That these may burst into the follicles and so appear at the surface is of course possible, but in the multitude of cases examined such an accidental communication has never been found. It is therefore highly probable that the causation of the pustular surface lesions is not to be met with in the glands; and, moreover, the absence of iodine from their contents in the great majority of cases is strongly against the premiss of such a relationship. So far as recent observations lead us, it seems safer to regard the skin manifestations as being due to vaso-motor neurosis induced in some unknown way by the action of the drug; and to this also the consensus of histological opinion points. The only constant changes on which all observers are agreed are, marked dilatation and sometimes thrombosis of the cutaneous blood-vessels, with alteration and partial destruction of the oil-glands; while, on the other hand, the sebaceous glands and hair follicles are only in isolated instances, and but to a limited extent, involved in the inflammation.

The *papulo-pustular* form usually begins as small shot-like papules which resemble somewhat the earliest stage of the variolar rash, a resemblance which is much increased as they become vesicular, when they also show a marked tendency towards umbilication. They quickly pass into a pustular stage, the pustules being more acuminated than those of the so-called bromic acne," and being seated on an erythematous and somewhat infiltrated base. The writer has seen the eruption resemble in a very marked degree the vesicular eruption of herpes zoster, and, like it, leave on drying up and the subsequent separation of the crusts, a small but distinct whitish scar. The seats of predilection are the face, the upper part of the thorax in front and behind, and, less frequently, the extremities. From these primitive forms all degrees of development may be met with. The spots may become confluent, resembling the romide eruptions, or may tend rather in a villous direction, the contents of the bullæ remaining clearer than in the analogous bromide eruption. In rare cases the confluent form is associated with epithelial proliferation and a resulting papillomatous upgrowth, and when to this is added the introduction of pyogenic organisms and the formation of pus, which discharges through multiple small openings, we have the condition described by Besnier as *acné anthracôïde iodo-potassique*." In a case described by Duhring "there was a confluence of the nodules, forming a sharply defined, rounded, inflammatory patch, violaceous in colour, its centre depressed and crusted, while at its periphery was studded with deep-seated, yellowish, sebaceous-looking pustules, presenting

an acneiform appearance." The *vesicular* and *bullous* forms are much less frequently met with; while the *urticarial* and *purpuric* varieties may be regarded solely as society show cases. The condition described by Hutchinson as "iodide hydrops" is simply an exaggerated form of the bullous eruption. A fatal case of the purpuric form occurring in a young child and following a dose of 2½ grains has been recorded by Stephen Mackenzie, and illustrated with a capital plate.

No fixed relationship in time can be laid down as existing between the administration of the drug and the appearance of cutaneous symptoms. It varies largely in different cases, and depends among other things on two main factors: firstly, the relative susceptibility of the individual; and, secondly, the amount of the drug administered. But it has been already pointed out that the latter bears very little relation to the occurrence of skin lesions, because, as has been observed, tolerance of large doses is sometimes seen in those who react promptly to the drug in a minimal dose. Nor has eliminative activity any except a secondary relationship; because it has been shown that, on the one hand, vascular or renal inadequacy presupposes a saturation which we know does not exist, and, on the other hand, that the cutaneous symptoms may not manifest themselves for a considerable time after the drug has been suspended. This feature of dosage has been explained on the ground that, when given in large quantity, iodide of potassium acts as a diuretic; while on withdrawal or reduction of quantity this eliminative activity ceases.

As regards *treatment*, what has been said under the bromide eruptions applies equally here. As a prophylactic measure the most valuable, beyond doubt, is the free dilution of the drug with large quantities of natural or artificial mineral waters, of milk, or of other diluents. The graver forms, besides stoppage of the drug, may require tonic and stimulant treatment, owing to the powerfully depressant effect it has in many cases.

*Iodoform* does not usually give rise to any skin manifestations when administered internally, or injected as an emulsion into the joints or subcutaneous tissues; yet in some cases the extension of the eruption spreads so far wide of the area of application that many dermatologists regard it as a constitutional intoxication quite apart from the local dermatitis caused by its direct application. This may be a punctiform, eczematous, or vesicular eruption, spreading with a rapidity and an advancing border of vivid redness almost indistinguishable from erysipelas. Here again idiosyncrasy counts for much; and the writer has seen one case of rodent cancer in which a second application of the powdered drug reproduced, after an interval of five years, with absolute exactitude the features of a previous attack due to the same cause, viz.

an acute vesicular erythema in the central parts with a peripheral vivid red and infiltrated border closely resembling erysipelas. In the diminishing use of iodoform since the practice of aseptic surgery has become more general these eruptions are less frequently seen, but for that very reason it is the more necessary for the practitioner to be familiar with their occurrence.

**Mercury.**—Since the introduction of antiseptics into the treatment of wounds every student has been familiar with the eruptions produced by the local action of various mercurial salts upon the skin. These are most commonly erythematous, vesicular, or pustular, or frequently a combination of all three. The vesicles are minute, extremely numerous, and almost always become pustular, especially when situated close to the pilo-sebaceous follicles of the skin. But nearly a century ago Alley called attention to a condition which he called *Hydrargyria*, following upon the internal administration of mercury, and which he grouped under three main headings as regarded type—the *mild*, the *febrile*, and the *malignant*. It may be noted here that the association between these eruptions and the internal use of the drug has been denied by one of the highest authorities, but the observations of by far the large majority of modern dermatologists amply confirm the older teaching. Almost any of the preparations of mercury may give rise to cutaneous symptoms, but by common consent the one most frequently found to produce them after internal administration is calomel. Although Alley's classification is not now generally adopted, most observers are agreed upon the occurrence of two well-defined forms—a *mild* and a *grave*. The former is simply an acute erythematous rash accompanied with intense itching and the formation of vesicles, and most frequently distributed on the inner surfaces of the thighs, the scrotum, the groin, and the lower part of the abdomen. The *grave* form is merely an exaggerated type of this, in which the vesicles become pustular or even bullous, associated with considerable fever and general malaise, and sometimes with acute inflammations of the related lymphatic glands. Desquamation follows as the patches dry up, and in rare cases repeated exfoliations may occur. It is highly probable that in the glandular cases pyogenic infection has been grafted on by the scratching of the inflamed and itching surface. Stoppage of the drug is generally followed by rapid subsidence of the symptoms, but where the irritation is great the application of calamine lotion or dusting the part with powdered starch and oxide of zinc affords great relief.

**Morphin** has long been known as having an irritant action on the skin, and *pruritus opi* was familiar to the earliest writers. When a rash is associated with the tingling and itching of the skin it is invariably erythematous, re-

sembling closely the eruption of scarlatina, but confined mainly to the face, neck, and the flexor aspects of the limbs, and preceded by a sensation of local heat and irritation. Desquamation to a slight degree follows as a rule. In rarer cases the erythema is more macular, and the resulting rash morbilliform in character. Suspension of the drug is the cure. It has been pointed out, however, that these cases, in which the administration of morphin has been followed by skin eruptions, show under all conditions a degree of marked susceptibility to the other influences of the alkaloid.

**Quinine**, or other preparations derived from cinchona bark, may give rise to eruptions either by external contact or by ingestion. The former may be typically seen among workers in quinine factories, and is usually confined to those parts which, either by exposure, or by the natural occurrence of folds or creases, offer the greatest facilities for the prolonged or continuous action of the irritant. These are, for example, the hands, wrists, flexures of joints, inner surfaces of thighs, and the genitals. The eruption is usually eczematous in character, and may be papular, vesicular, or pustular, while in aggravated cases pemphigoid forms are met with, associated with the formation of extensive scales and crusts which leave frequently on separation red and oedematous or oozing surfaces. Removal of the irritant and the local application of calamine or subacetate of lead lotion, with the addition of liquor carbonis detergens (Ziss ad 5), rapidly allay the discomfort and promote the cure. It has been observed, however, that no immunity is acquired by exposure, and the individual is always liable to a recurrence on resumption of the offending occupation.

The eruptions following the internal administration of quinine are essentially polymorphous, varying from the most elementary to the most complicated forms of cutaneous lesion. Thus erythematous, urticarial, papular and vesicular, petechial, bullous, and gangrenous forms have been described (Morrow). In the large majority of cases—almost two-thirds—the rash is scarlatiniform, and hence its clinical significance. It is of a bright, vivid redness, completely disappearing on pressure, and showing itself first in the face and neck, but rapidly becoming generalised over the whole surface of the body. There is generally considerable, and sometimes severe, congestion of the mucous covering of the palate and fauces, and when the onset of the eruption is associated with high fever (as in a few recorded cases), and followed by a desquamation lasting from a few days to several weeks, the mimicry is complete, and the diagnosis from scarlatina is a matter of great difficulty and doubt for prophylactic reasons. The absence of high fever, as a rule, and of precedent gastric disturbances is generally conclusive, but the demonstration of quinine in



the urine is in many cases the ultimate and only absolute test (*note "Urine"*) Stoppage of the drug effects a rapid cure

**Salicylic Acid and Salicylates**—The cutaneous lesions produced by the external use of salicylic acid as an antiseptic have long been familiar to surgeons, and assume generally the form of a vesicular erythema which is always limited to the area with which the agent is brought into contact. More variable results follow its internal administration, either as the acid or as the sodium salt, and erythematous, vesicular, urticarial, and hæmorrhagic varieties have been recorded. The erythematous is the commonest form, and may be either localised or general. It is frequently associated with oedema, especially affecting the face (eyelids and lips) and the extremities, a certain amount of perspiration, and frequently a rise of temperature,—manifestations which bear a striking resemblance to those produced by atropin, belladonna, and chloral, and probably associated with a similar causation, vaso-motor disturbance. No special clinical importance is attached to it, and the stoppage of the drug invariably puts an end to the associated cutaneous phenomena. The presence of the acid is easily recognised in the urine by the ferric chloride test.

**Sulphonil**, in doses varying from seven and a half to thirty grains (0.5-2.0 gms.), has been followed by a bright erythematous and scarlatiniform eruption attended with intense itching, occurring chiefly on the thorax, shoulders, and thighs, and followed, after its subsidence in a few days, by slight desquamation. It is of no special import.

**Tar**, and its derivative *Carbolic acid*, produce, by direct contact with the skin, certain lesions which may vary from a simple eczematous eruption in the weaker forms to complete destruction of tissue by the stronger, as by the application of pure carbolic acid. Externally, continuous irritation with tar, such as occurs in gas works, produces the so-called "tar acne," which is caused by the plugging of the orifices of the pilo-sebaceous follicles during contact with the material, and the formation subsequently of papules or nodules as the result of the inflammatory changes in the fibrous tissue surrounding the duct. These nodules may break down and ulcerate, the process being associated with increased epithelial activity which in time may transgress the bounds and become malignant, developing into what is known as "tar cancer" (epithelioma). Though doubt has been thrown on this occurrence, too many cases have been recorded to leave any room for doubt as to its reality, and several have come under the writer's personal observation. In the earlier days of Lister's moist carbolic dressings it was almost the rule, rather than the exception, to see a localised vesicular eczema produced in and about the area protected under the waterproof

covering. Similar effects have been observed to follow the injection of carbolised fluids into mucous and serous cavities. In other cases erythematous and eczematous outbreaks have followed the absorption of the drug. When poisoning in moderate degree occurs from absorption, the urine becomes of a dark olive-greenish colour on standing, and the presence of carbolic acid can be demonstrated by appropriate tests. Treatment, of course, consists in removal of the cause. When symptoms of general intoxication are acute, diuretics and large doses of sulphate of soda should be given internally, and active measures taken to combat the associated collapse.

**General Principles of Treatment**.—But little remains to be added to what has been incidentally said above as to the main indication in treatment.—It is, in all cases, stop the drug or diminish the dose. Many patients who cannot bear a drug in one form will take it readily in another, and here again nothing but careful observation and repeated trial can help the practitioner. As a general rule, it may be laid down that *all drugs liable to produce toxic eruptions should never be given in a concentrated form, but always in the maximum degree of dilution*. In this way we know we are nearest to the border of safety. As to the so-called "preventive" additions—e.g. arsenic and ammonia to bromides and iodides, hydriodic acid to quinine, etc.—opinions will always differ, and individual experience will lead to perfectly sound but very divergent opinions. Above all, the point of importance is to recognise the relation between a given drug and a known eruption, and that being ascertained, treatment resolves itself into the alphabet of therapeutics.

#### ANTITOXIN RASHES

**Causation**—In a very large percentage (20 to 45 per cent) of cases treated with antitoxin, rashes are apt to occur at a variable period after the injection. The antitoxic principle employed has nothing to do with their causation, nor are they due to any antiseptic which may be used in the preservation of the serum. They depend entirely on the serum itself, as is proved by the fact that normal horse serum produces them. They appear less frequently with the concentrated preparations now in vogue than they used to do when the injection of large amounts of a dilute antitoxin was common. They are also less likely to follow the administration of one large dose than they are the injection of several successive and comparatively small doses.

**Occurrence and Duration**—The rash may occur any time between the first and twenty-fourth day after the injection. A series of cases treated with small doses of dilute antitoxins in the Edinburgh City Hospital gave an average occurrence at about the twelfth day. More recently, however, the concentrated preparations now in use have appeared to cause an

earlier appearance of the eruption, and rashes on the third, fourth, and fifth days are very common.

The rash may last from a few hours to about a fortnight. As a rule, however, its duration is not more than five days, and many disappear on the second, third, or fourth day.

In certain instances, probably when the blood of two or more horses has been mixed in preparing the antitoxin, successive rashes, different in character, may appear at different dates.

**Character.**—Generally speaking, the character of the rashes is "septic," that is to say, they are of that kind which we are accustomed to associate with blood-poisoning from various causes. They are always *erythematous*, and occasionally may be classed as true *urticaria*. The commonest form is that of an *erythema multiforme*, a bright pink eruption, macular or papular in character, with a tendency in many instances to circinate arrangement, and often showing an eccentric extension of the individual macules or papules. Its distribution is very variable, as it may appear on any part of the body or face, usually being most marked in the neighbourhood of the larger joints and at the seat of injection. It also appears to have a certain preference for the extensor surfaces. Occasionally it may be morbilliform or scarlatiniform in character, but neither of these forms is very common. In many cases it is exceedingly itchy, and it is this characteristic which is most distressing to the patient, who otherwise often feels quite well. When the eruption is *urticarial* it is marked by the usual wheals characteristic of that condition, and these frequently occur on the face.

**Other symptoms.**—As regards *pyrexia* some cases show no fever at all, and others only slight deviation from the normal. On the other hand, in young children especially, there may be a considerable rise, the chart showing temperatures of 101° to 103° even when there is no complicating arthritis.

**Edema** is not uncommon, and in some instances may involve considerable areas of skin, though more frequently it is limited to the hands and feet.

**Arthritis** is a very common complication, and indeed may occur without a rash at all. The joints of the extremities are most frequently complained of, and may be swollen and tender.

**Diagnosis.**—There is not usually any difficulty in diagnosing the rash. It is to be looked for in a large proportion of cases injected, and therefore its occurrence need cause no surprise. Occasionally, however, when scarlatiniform or morbilliform in character it may cause anxiety. The marked itchiness of the serum rash, the absence of catarrhal symptoms (in its measles form), and of those of the usual invasion of scarlatina, should decide the case.

**Treatment.**—The rashes scarcely require any treatment, and their presence has little or no

clinical significance. To relieve the itching, however, tepid sponges or the application of a lead lotion may be found useful. For the arthritis, salol and the salicylates often appear to give relief. Some prefer a palliative treatment, with laudanum fomentations and opium internally. The exceedingly variable duration of untreated cases makes it almost impossible to judge the efficacy of any treatment.

The following Plates will be found useful for reference—

- Argyria—Crocker's *Atlas*, Pl. XXXIII.  
 Arsenic—Cancer, *Arch. of Surg.* Pl. IX.  
 " Keratosis, " Pl. XVII.  
 " Psoriasis, " Pl. XIV.  
 " Pigmentation } Crocker, Pl. XXXIII.  
                             & Keratosis,  
 Bromide—Confluent form, " Pl. XXXV.  
 " Ulcerative, *Syd. Soc. Atl.* Pl. XLIV.  
 " Bullous, *Ill. Med. News*, Jan. 1889.  
 Chloral—Erythema, *Arch. of Surg.* Pl. V.  
 Chrysarobin—Erythema, Crocker, Pl. XXXVIII.  
 Iodide—Bullous, " Pl. XXXVI.  
 " { Tubercous, *Arch. of Surg.* Pl. III, IV.  
       { (fatal case)  
 " { Purpuric, *Ill. Med. News*, Nov. 1888.  
       { (Mackenzie's case)  
 " Bullous, *Syd. Soc. Atl.* Pl. XXXIII.

**Drug Habits.** See ALBINTHISM, ALCOHOLISM, ANAESTHETICS, ARSENIC, CANNABIS INDICA, CHLORAL, CHLOROFORM, COCAINE, ETHER, HYPNOTICS, HYPNOTISM (*Uses*), MORPHINOMANIA AND ALLIED DRUG HABITS (*Antipyrin, Antifebrin, Paraldehyde, Chlorodyne, etc.*), SULPHONAL, TRIONAL, VERONAL, VICE (*Drug Habits and Smoking*).

**Drummond-Morison Operation.**—An operative method of treating ascites introduced into use by Dr. David Drummond and Mr. Rutherford Morison in 1896, its object is to develop vascular anastomoses between the portal and systemic venous systems. As performed by Drummond and Morison, the abdomen was opened and dried out with sponges, the parietal peritoneum and that covering the liver and spleen were scrubbed with a sponge; the omentum was sutured across the anterior wall, and a tube was left in the pouch of Douglas. The ascites was cured, and Talma and Schüssler and others have since obtained equally satisfactory results by means of the same or slightly modified methods, so that, in 1905, Bungo was able to collect 274 cases from literature (including 14 of his own) in which the operation had been performed. The indication for the procedure is portal obstruction from hepatic cirrhosis, but it is applicable in portal obstruction due to any cause, the most marked benefit following it is relief of the ascites, but hæmorrhages from mucous membranes (e.g. hæmatemesis) may also be greatly benefited,

in about 30 per cent of the recorded cases the ascites was relieved

**Drunkenness.** See ALCOHOLISM.

**Dry Labour.** See LABOUR, FAULTS IN THE PASSENGER (*Early Escape of Liquor Amni, Dry Labour*).

**Dry Mouth.** See SALIVARY GLANDS, DISORDERS OF (*Xerostomia*)

**Dry Rot.**—Timber (*eg* in the construction of a house) which is subject to alternate wetting and drying, or which is imperfectly ventilated, decays, it is affected either by wet or by dry rot (*Merulius tachymans*), diseases due to the growth of the mycelium of fungi and in which the wood is reduced to a powder, such wood must be entirely removed if the process is to be stopped, preventives are found in thorough ventilation of beams and joists, by painting and varnishing, and by forcing creosote into the wood under pressure (Bethell's process)

**Dual Personality.** See MEMORY IN HEALTH AND DISEASE (*Periodic Complete Loss of Memory*), DOUBLE CONSCIOUSNESS, UNCONSCIOUSNESS (*Double Consciousness*)

**Dubini's Disease.** See CHOREA (*Electrical Chorea*), SPASM (*Varieties, Paramyoclonus Multiplex, Diagnosis*)

**Duboisine.**—An alkaloid, probably identical with hyoscyamine ( $C_{17}H_{21}NO_4$ ), it is obtained from the leaves of *Duboisia myoporoides*, and is found in *Datura Stramonium* and *Hyoscyamus niger*, it resembles atropine in its action, *eg* it is a mydriatic, but its effects as such are more quickly produced and more evanescent, its toxic effects also differ from those of atropine. It has been used as a mydriatic (as ophthalmic discs containing 1/500 grain), and in exophthalmic goitre. See ALKALOIDS, TOXICOLOGY (*Alkaloids, Henbane and Stramonium*)

**Duchenne-Erb Paralysis.**—A type of paralysis in which the deltoid, biceps, brachialis anticus, and supinator longus are affected. See TRAUMA, DANGEROUS (*Lead-Poisoning*), BRACHIAL PLEXUS, SURGICAL AFFECTIONS OF.

**Ducrey's Bacillus.**—Ducrey's bacillus of soft sore, a micro-organism causing acute suppuration. See SUPPURATION (*Etiology*), VENEREAL DISEASE (*Soft Sore or Chancroid*)

**Duct or Ductus.**—A canal, usually serving the purpose of conveying the secretion away from a gland, sometimes acting as a communication between one blood-vessel and another. Instances of the former are found in the *bile*-ducts, the *cystic duct*, *Bartholin's*, *Gartner's*, *Muller's*, *Wharton's*, *Stenson's*, and *Wirsung's* duct; and of the latter in the *ductus*

*arteriosus*, the *ductus venosus*, the *thoracic duct*, and the *ducts of Cuvier*

**Ductless Glands.** See PHYSIOLOGY, INTERNAL SECRETIONS (*Suprarenal Bodies, Pituitary, Thyroid, Ovaries, Thymus, etc*)

**Ductus Arteriosus.** See HEART, PHYSIOLOGY OF (*Embryology*), HEART, CONGENITAL MALFORMATIONS OF (*Patent Ductus Arteriosus*), PHYSIOLOGY, REPRODUCTION (*Fetal Circulation*)

**Ductus Venosus.** See HEART, PHYSIOLOGY (*Embryology*), PHYSIOLOGY, REPRODUCTION (*Fetal Circulation*)

**Duga's Test.**—A test for dislocation of the shoulder, if, when the hand of the dislocated side is placed on the opposite shoulder, the elbow is elevated and stands out prominently in front of the chest, there is really a dislocation. See SHOULDER, DISEASES AND INJURIES OF (*Dislocations, Signs and Symptoms*)

**Duhring's Disease.** See DERMATITIS HERPETHIFORMIS

**Duhrssen's Operation.**—A method of vaginal fixation of the uterus in cases of obstinate retroversion

**Dulcamara.**—The branches of *Solanum dulcamara* (Bittersweet or Woody Nightshade), containing two alkaloids (solanine and dulcamarine,  $C_{24}H_{40}O_{10}$ ), used as an alternative in some skin diseases, in chronic rheumatism, in whooping-cough, etc., not now official in the British Pharmacopœia

**Dulcite.**—A polyhydric (hexahydric) alcohol, isomeric with mannite ( $C_6H_{14}O_6$  or  $C_6H_8(OH)_6$ ), it is used to sweeten the food in the treatment of diabetes

**Dulness.** See PLEURA, DISEASES OF (*Acute Pleurisy, Signs of Pleural Effusion, Percussion*), ABDOMEN, CLINICAL INVESTIGATION OF (*Percussion*), CHEST, CLINICAL INVESTIGATION OF (*Percussion*), etc

**Dumb Ague.**—Masked intermittent fever, masked malaria

**Dumbness.** See DEAFMUTISM, CRETINISM, HYPNOTISM (*Psychical Dumbness*), MENTAL DEFICIENCY, etc

**Dum-Dum Fever.**—Piroplasmiasis, or kala-azar, or tropical splenomegaly, a chronic disease of India, China, Egypt, and the Tropics, in which the spleen and liver are enlarged, there are hæmorrhages and transitory oedemas, a moderate degree of anæmia, and irregularly remittent fever, a protozoon parasite (*Donovan-Leishman bodies*) has been found in the spleen; quinine is useful, but has not the same good effect as in malaria.

**Duodenitis.**—Inflammation of the duodenum (q.v.).

**Duodeno-**.—In compound words *duodeno-* means relating to the duodenum e.g. *duodenotomy* is the operation of cutting into the duodenum, *duodenostomy* is that of making a permanent opening into the duodenum through the abdominal wall, *duodeno-enterostomy* is that of making a permanent communication between the duodenum and another part of the intestine, and *duodenocholecystostomy* is that of making a permanent communication between the duodenum and the gall-bladder.

**Duodenum.** See also ABDOMEN, INJURIES OF (*Lesions of Intestine*), APPENDICITIS (*Diagnosis from Duodenal Ulcer*), BURNS AND SCALDS (*Intestinal Complications, Ulcer of Duodenum*), HEMATEMESIS (*Causes and Source*), INTESTINES, DISEASES OF (*Malformations of the Duodenum*), INTESTINES, DISEASES OF (*Ulcers of the Duodenum*), INTESTINES, DISEASES OF (*Malignant Disease*), LIVER, DISEASES OF (*Aneurysm of Hepatic Artery, Diagnosis*), MELFNA (*Causes, Duodenal Ulcer*), PERITONITIS, ACUTE GENERAL (*Etiology, Perforation of Duodenal Ulcer*), PHYSIOLOGY, FOOD AND DIGESTION (*Alimentary Canal, Duodenum*), POST-MORTEM METHODS (*Examination of the Body-Cavities*), STOMACH AND DUODENUM, DISEASES OF (*Anatomy, Digestion, Diseases, Morbid Anatomy, Ulcer, etc.*), STOMACH, SURGICAL AFFECTIONS (*Complications of Gastric Ulcer*)—Affections of the duodenum are of great interest both from a medical and surgical stand-point. But, as the great majority of diseases which involve the duodenum cannot be clearly differentiated clinically from those of the pyloric end of the stomach and the small intestine, these will be described in other articles. The object here is to present a brief epitome of the various disorders.

There are certain points of anatomical and physiological importance, the position, the horse-shoe shape, the close connection with the head of the pancreas, and the entrance of the common bile duct, are all of great clinical importance.

With regard to position this may be slightly modified by alterations in the position of the stomach and intestine (*vide* "Enteroptosis") Short of enteroptosis, however, a careful examination of post-mortem cases made at random readily reveals greater variations in the position of the abdominal viscera than is usually thought of during life.

The close relationship between the receptive and motor mechanism of the duodenum and the stomach, and also the influence of its secretion, is fully considered in the article "Digestion and Metabolism."

The most striking and obvious perversion of this mechanism is seen in the familiar class of case known as bilious vomiting, where, as a

result of combined motor hyperactivity and relaxation of the pyloric orifice, the vomiting of bile is brought about. Apart from this obvious derangement, it is likely that there are many other perversions of function that are not so readily nor so definitely represented by definite outward symptoms. Thus it is impossible to believe that in many cases of dyspepsia the function of the duodenum is not very definitely interfered with. We are led to this opinion by, among other things, the close relationship that obtains between the motor activity of the muscle of the duodenum and that of the stomach and intestine. In the absence of obvious changes in the feces, we are perhaps not fully justified in making a diagnosis of *duodenal dyspepsia*, but it is not unlikely that some cases of dyspepsia, where the various sensory symptoms develop several hours after the ingestion of food, associated it may be with the presence of pain on pressure to the right of the middle line in the right hypochondriac region, are really cases where the secretory or sensori-motor mechanism of the duodenum is primarily at fault.

A simple ulcer is met with here which may present all the clinical features of, and be clinically indistinguishable from, gastric ulcer. The ideas formerly entertained regarding the great frequency of ulceration of the duodenum as a sequel of burns have been modified somewhat during the last few years, merely as a result of more careful investigation into the truth of the old text-book statements (*see* "Burns").

Simple ulceration may develop as a result of irritation from a large gall-stone.

**Malignant Disease**—This is by far the most important lesion, and specially in view of the possibility of beneficial results following early operative interference. As it is impossible clinically to differentiate between a primary morbid growth of the duodenum and one involving the head of the pancreas or pylorus, this condition will be described more appropriately in the section on these viscera. It is sufficient to indicate here the great importance of a thorough examination of the feces as a diagnostic factor in such cases. The absence of bile in the stools suggests some obstruction to the excretion of bile probably involving the orifice of the duct, the presence of undigested fat in the feces, and occasionally the presence of glycosuria, would rather point to the pancreas as the primary focus of disease.

**Inflammations**—These are of no great clinical significance in themselves, they may be secondary to gastric catarrh, or merely a local manifestation of the general enteritis present. If severe, jaundice may develop, this, however, usually signifies an extension of the catarrhal process to the bile-ducts (*see* "Gall-Bladder and Bile-Ducts").

**Dupuytren's Contraction.** See DEFORMITIES (*Hand and Fingers, Congenital Contraction, Diagnosis*), FASCIA (*Contraction of the Palmar Fascia*), GOUT (*Morbid Anatomy, Urates of Soda in Tendon Sheaths*).

**Dupuytren's Fracture.** See ANKLE-JOINT, REGION OF, INJURIES (*Fractures in the Vicinity of*).

**Dura Mater.**—The outer membrane, strong and fibrous (*dura*), which covers the brain and spinal cord (cerebral and spinal dura). See INSANITY, PATHOLOGY OF (*Pathological Anatomy, Dura Mater*), MENINGES OF THE CEREBRUM (*Anatomy, Vascular Disturbances, Inflammations, Tumors*, etc.).

**Durande's Mixture.**—A mixture of ether and turpentine given in the hope of dissolving gall-stones.

**Duret's Balsam.**—An antiseptic ointment (*brume Duret*), said to contain coal-tar, oil of cade, resinoin, menthol, guaiacol, camphor, sulphur, borax, glycerine, acetone, castor oil, and wood oil, it is, therefore, a very complex preparation, containing nine antiseptics, some of which are also mild cutaneous stimulants (*vide Lancet*, June 16, 1906).

**Duroziez's Sign.**—The double murmur which can sometimes be produced by pressure on one of the large arteries (e.g. the carotid) in cases of aortic incompetence.

**Dust Diseases.**—Diseases due to the inhalation of particles of dust (*pneumonokonoses*), such as coal dust (*anthracosis*), metallic (steel) dust (*siderosis*), and stone dust (*chaconosis*), tuberculosis and typhoid fever may be spread also by infected dust particles. See LUNGS, PNEUMOKONOSIS, TUBERCULOSIS (*Portals of Invasion and Channels of Spread*), TYPHOID FEVER (*Etiology*).

**Dutton's Disease.**—Human typhus-somnolence. See PARASITES (*Protozoa*).

**Dwarfism.** See also ETHNOLOGY (*Dwarfs Races*), ATROPHY, HYPERTROPHY, ACHONDROPLASIA; LABOUR, PROLONGED (*Pelvic Deformities*), MICROSMIA, etc.—The word "dwarf," a very old term, has in its progress down the centuries lost much of its significance and of the sharpness of definition which it may once have had. It now signifies simply a diminutive adult human being, and includes cases of stunted stature due to postnatal diseases of the spine and lower limbs (rickets, etc.), as well as to antenatal maladies (achondroplasia) and to teratological states. "True dwarfism" is an expression which has been introduced in order to define more exactly the conditions which alone ought to be embraced under the name a true dwarf is a person

of unusually small stature, not the result of any particular disease or deformity, but merely from growth having fallen much short of the usual standard. In this restricted sense the word dwarfism is practically synonymous with *microsmia*, and in this article it is true dwarfism or *microsmia* that is being considered.

True dwarfism or *microsmia*—a teratological smallness of all the parts—may be antenatal or postnatal in origin. There is, for instance, embryonic *microsmia*. Sometimes when we are engaged in examining an abortion sac we find to our surprise that it contains an embryo of a size much smaller than the size of the sac had led us to expect. Yet this embryo may show a degree of development in accordance, not with its size, but with that of the sac containing it and with the estimated age of the pregnancy. It may, therefore, be regarded as a "dwarf embryo," or a case of "embryonic *microsmia*", although it has, perhaps, been customary to look upon it as an example of early hydranmios or embryonic hydranmios. During foetal life, also, dwarfism may show itself (*foetal microsmia*). When, for instance, a fœtus is born at the full term, having a weight of less than 3 lbs. and a length of less than 12 inches, but possessing the other signs of maturity (finger-nails projecting, absence of lanugo, etc.), it is to be regarded as a case of foetal *microsmia* or foetal dwarfism. In Sir Everard Home's case, a female child weighing 1 lb. and measuring 7 inches was born at the full term (the mother had had a fright when three months pregnant), the child, known as the "Sicilian dwarf," died at the age of nine years, when she was nearly 20 inches in height, and had the ossification, dentition, and mental powers of a child of two rather than of nine years. There was, therefore, an arrest of both growth and postnatal development here, and to this combination it has been proposed by Hastings Gilford to give the name of "ateleiosis." Other instances of foetal *microsmia* were those of Nicholas Ferry or "Bébé" (weight at birth less than 1 lb., length 8 inches), "Princess Paulina" (12 inches in length when born), the "Marquis Wolga" (9 inches in length at birth and less than 2 lbs. in weight), etc.

Dwarfism, however, is by no means constantly present at birth: the retardation of growth may begin at one, two, three, or four years of age, indeed it may not commence until early adolescence. Jeffery Hudson, for instance, was not a congenital dwarf, and his parents were of average height, yet at the age of nine years he was only 18 inches high. He had some chequered experiences in an adventurous life: he was presented "in a pie" to King Charles I.; he fought a successful duel, for, as a writer says describing him, "although a dwarf, Jeffery was no dastard", and he was sold as a slave in Barbary. This last and most trying adventure had a special interest, for it caused, or at least

preceded, the recommencement of growth at the age of thirty years. Joseph Boruwlaski was born in Polish Galicia in 1739, and he died at Durham in 1837, at the great age of ninety-eight; he was never more than 39 inches in height, and he had a sister who measured 2 feet 4 inches, a brother who measured 41 inches, and another brother whose height was 6 feet 4 inches. This association of microsomia with macrosomia in the same family history is not uncommon. Some dwarfs have been above the average size at birth, this was the case with the famous "General Tom Thumb." In an observation reported by Claude, each alternate child in a family of eight was a dwarf.

Many causes of microsomia have been alleged, among which may be named poor or bad food, the infectious fevers, injuries (especially of the head), chronic hydrocephalus, idiocy, fetal rickets, alcoholism of the parents, and consanguinity or advanced age of the parents. Of course, if an antenatal cause be admitted, it must be supposed to remain latent for some time in the cases in which the dwarfism does not appear until later childhood.

Anomalies in development (malformations) are not uncommonly associated with dwarfism. Thus, dentition may be incomplete or retarded, the general ossification of the skeleton may be tardy, the skin may be hairless and the fingers almost nailless, the reproductive organs may be defective, as shown by sterility and want of sexual instinct in both sexes, by cryptorchidism in the male, and by absence of pubic hair and delayed menstruation or oligomenorrhœa in the female, and the mental powers may be of a low order, pointing to incomplete development of the higher nerve centres. Of course, exceptions to all these statements exist: some dwarfs, for instance, have been perfectly well formed, some have been able to have children, generally of normal size, some have lived to a good age, and some have had fair intellectual powers. It is evident, therefore, that the two processes which are going on side by side in antenatal life as well as in infancy (I refer to development and growth) do not necessarily fail together or succeed together. In other words, an infant may grow to its proper size although it is malformed or defective, and a child may be perfectly formed but a dwarf in size. At the same time it is not uncommon to note that both growth and development have suffered, and that the child that is stunted in size shows also defects in development (malformations) and in functional activity. A possible explanation of these irregularities may be found in the date when the arresting cause (whatever it may be) comes into action, for if it be effective in antenatal life it will more probably affect both development (which is then active) and growth, whereas if it only begin to show itself in childhood (when the

greatest number of the developmental processes are completed) it will delay or stop growth alone. What this arresting cause is has not yet been discovered, but it may be one or many, and it may be the same or (more likely) different for the different epochs of life (embryonic, fetal, infantile, adolescent). Possibly an internal secretion may be the root-cause of dwarfism, perhaps the thymus, the thyroid, the ovaries and testicles, and the pituitary body may all at one time or another in life preside over the processes of growth.

**Dwellings.**—In sanitary law a dwelling-house means "any inhabited building, and includes any yard, garden, out-houses, and appurtenances belonging thereto, or usually enjoyed therewith, and includes the site of the dwelling-house so defined" (*Housing of the Working Classes Act, 1890*). See CELLARS; CUNTIAGE, DISINFECTANT, SEWAGE AND DRAINAGE, VENTILATION AND WARMING, etc.

**Dyad.**—A bivalent element (e.g. calcium) or radicle (e.g.  $\text{SO}_4$ ) in chemical nomenclature.

**Dyaster Stage.** See PHYSIOLOGY, THE CELL. (*Mitosis*)

**Dying Declaration.** See MEDICINE, FORENSIC (*Dying Declarations*)

**Dymal.**—A non-irritating, antiseptic dusting-powder, drylyum salicylate.

**Dynamite.** See NITROGLYCERINE.—Dynamite is a mixture of nitroglycerine (75 per cent) and an infusional earth or kieselsol (25 per cent), the latter serves as an inert base, being composed chiefly of the fossil remains of diatoms. Another form of dynamite is that in which the nitroglycerine is combined with a combustible or explosive base, such as gun-cotton or potassium nitrate. Both the explosion and burning of dynamite give rise to obnoxious fumes which may cause poisoning, it also causes toxicological effects if swallowed. See TRADES, DANGEROUS (*Risks attending the Manufacture and Use of Explosives*)

**Dynamometer.**—An instrument for measuring the power of muscular contractions, especially the strength of a hand-grasp, by means of it a difference in the power of the muscles of the opposite sides of the body can be detected, e.g. in hemiplegia.

**Dyne.**—The absolute unit of force in dynamics, defined as the force which, acting on a mass of one gramme, will propel it with a velocity of one centimetre in one second.

**Dys-**—In compound words *dys-* (Gr.  $\delta\upsilon\sigma$ , meaning bad, ill, or hard) "destroys the good sense of the word or increases its bad sense"; thus *dysarthria* means defective articulation in speaking, *dysarthrosis* means a defective or dislocated joint, *dysbulia* means morbid impair-

ment of the will, *dyscholia* means a morbid condition of the bile, *dyscoria* means an irregular shape of the pupil, *dysmesia* means difficult vomiting, etc. etc

**Dysacusis.**—When an unpleasant sensation is caused by ordinary sounds (e.g. headache) it has been termed *dysacusis*

**Dysæsthesia.**—A morbid or perverted sensation (tingling, formication, tinnitus, etc.), or an impairment of sensibility, paræsthesia, the painfulness of a sensation which normally is accompanied by no pain. See *HYSTERIA (Sensory Disorders)*

**Dysanagnosia.**—Inability to read correctly from loss of power of understanding written signs, alexia or dyslexia, word-blindness

**Dysbasia Anglo-Sclerotica of Erb.**—Intermittent lameness or claudication (*g.v.*), or Walton's crural angina, it is found in cases of aneurysm of the iliac artery and in arterio-sclerosis, and it is due to diminution in the supply of blood to the muscles

**Dyschezia.**—Pain or difficulty during defecation. See *LABOUR, INJURIES TO THE GENERATIVE ORGANS (Evidence of Injuries to the Pelvic Articulations, Sacro-Coccygeal Joint)*

**Dyschromatopsia.**—Incomplete colour-blindness or diminished colour-sense. See *COLOUR VISION (Congenital Colour-Blindness, Varieties)*

**Dyscinesia.**—Diminished or impaired power of voluntary movement. *Uterine dyscinesia* is the name which has been given to pain produced by walking and other ordinary movements, and referred to the uterus (Graily Hewitt).

**Dyscrasia.**—A faulty state of the constitution or of the blood (as the result of such a disease as cancer), the word literally means an abnormal or bad mixture (from Gr *δυσ-*, and *κράσις*, a mixture), cachexia, the *hemorrhagic dyscrasia* is hemophilia, the *uric acid dyscrasia* is the uric acid diathesis, *dyscrasia saturnina* is lead cachexia, etc

**Dysdiachoresis.**—Constipation (from Gr *δυσ-*, hard, and *διαχρᾶειν*, to pass through)

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See also *COLON, DISEASES OF (Simple Colitis, Diagonosis), ENEMATA (Diarrhoea and Dysentery), FÆCES (in Dysentery), LIVER (Acute Hepatitis), LIVER (Abscess, Tropical), LIVER (Portal Thrombosis), LUNG, ABSCESS OF (Amœbic Dysentery), MALARIA (Dysenteric Form), NERVES, MULTIPLE PERIPHERAL NEURITIS (Cause), TYPHOID FEVER (Sequelæ), WATER (Disease produced by Contaminated Water)*

SYNONYMS Gr *Δυσεντερία*, L. *Tormina*, E. *The Bloody Flux*, G. *Ruhr*, F. *Dysenterie*.

**DEFINITION.**—Dysentery is a clinical term connoting a complex of symptoms dependent on inflammatory, ulcerative, and gangrenous lesions of the large intestine. It may be roughly defined as a group of closely allied infective diseases, characterised by frequent mucous, bloody, or serous stools, by griping pains (*tenesmus*), more or less straining (*tenesmus*), generally with retention of feces.

**BACTERIOLOGY.**—In certain cases of dysentery bacteria alone are met with in the stools and intestinal lesions, in others, amœbæ as well as bacteria are present. The precise significance of the amœbæ in relation to the dysenteric process has not been fully determined, but as they are associated with a form of the disease presenting distinctive clinical and pathological characters, we are justified in admitting a non-amœbic and an amœbic form of dysentery. The diversities observed in the clinical features and in the lesions of non-amœbic dysentery, taken along with the results of bacteriological researches, indicate that this form will ultimately be found to comprise two or more distinct varieties.

In non-amœbic and amœbic dysentery alike, the common bacteria of suppuration, staphylococci and streptococci, are met with in the stools, as, indeed, they frequently are in the stools of healthy persons. The staphylococci, so far as is known, are not in any sense specific, but when the disease is once established they doubtless bear a part—perhaps by no means an inconsiderable one—in promoting suppuration and destruction of tissue. The three varieties of the staphylococci—anærus, albus, and citreus—are present in the stools, and are found with equal frequency in the epidemic dysentery of temperate climates and in the chronic dysentery of tropical regions. Strepto-

ococi are often very abundant in dysenteric stools, their multiplication being favoured by the inflamed condition of the bowel. All pathogenic micro-organisms vary in virulence, but none more so than the streptococci, and experiments show that in some forms of dysentery, at least, they play an important part in the dysenteric process. The experiments of Zancarol and of Celli and Fiocca seem to place it beyond doubt that dysenteric lesions may be produced by the administration, by the mouth or rectum, of pure cultures of virulent streptococci derived from dysenteric stools. While the former pathologist inclines to regard the streptococcus as the principal pathogenic agent in dysentery, Celli and Fiocca relegate it to a secondary position, but claim for it the power of exalting the virulence of the bacterium coli commune (which they look upon as the ordinary specific microbe of dysentery) into that variety which they have named the *bacterium coli dysentericum*. These pathologists have occasionally found in dysenteric stools a small species of proteus which is also found to intensify the virulence of the *B. coli* commune, and with which they have succeeded in some instances in producing dysenteric symptoms in animals.

Bertrand and Baucher found the *B. pyocyaneus* in the stools both of the epidemic dysentery of France and those of the chronic dysentery of warm climates, but more abundantly in the former. Calmette found this organism not only in the stools of acute dysentery in Cochin China, but also in the ulcerations of the large intestines and in the blood. He ascribes to it the preponderating rôle in the causation of dysentery, maintaining that it alone of the microbes present in the stools is capable of reproducing the lesions of dysentery. These statements have not been confirmed.

Anaerobic vibrios are met with in a considerable number of cases both of the acute and chronic disease. The inflamed condition of the stools is ascribed to the presence of these microbes. They are merely occasional and subsidiary agents.

All recent observers recognise the bacillus coli communis, or rather a virulent variety of it, or some closely allied organism, as one of the most important pathogenic agents of dysentery. It was one of the microbes constantly met with by Bertrand and Baucher. This was probably the bacillus found by Chantemesse and Vidal in the walls of the intestine and mesenteric glands of one who had died of dysentery, and in the stools of those suffering from the disease. Maggiora found it in large numbers in every case he examined, and he proved experimentally that it could produce dysenteric lesions in animals. It is to the researches of Celli and Fiocca, however, that we are chiefly indebted for our knowledge of the part played by this bacillus in the causation of dysentery. They,

in collaboration with others, have shown that by itself it is capable of setting up dysentery in animals whether administered by mouth or rectum, and that the toxins obtained from pure cultivations give rise to dysenteric symptoms and lesions, that the serum of dysenteric patients causes agglutination of the *B. coli dysentericum*. This bacillus, although capable by itself of giving rise to dysentery, is generally associated with streptococci, which exalt its virulence within the body and in faecally polluted soil. Ainaud, according to Scheube, has also come to the conclusion that this bacillus, when its virulence has become intensified by association with other microbes, or in some other manner, is the specific microbe of dysentery.

Quite recently Shiga, working in Kitasato's Institute in Japan, has isolated and cultivated a bacillus which is not found in healthy men or animals, but which is always present in dysenteric stools, in the lesions of the colon and rectum, and often in the mesenteric and retro-peritoneal glands of those who have died of dysentery, but never in the liver or spleen. He describes it as a short bacillus, similar in morphological characters to the colon bacillus. He found the cultures of this microbe to present the phenomenon of agglutination with the serum of persons suffering from dysentery, but not with that of healthy persons or of those suffering from other diseases. This bacillus is believed to be identical with the *B. coli dysentericum* of Celli and Fiocca. There is thus a large amount of evidence pointing to a variety of the *B. coli* communis as one of the chief agents of epidemic dysentery, and possibly also of other forms of the disease.

The Johns Hopkins Commission to the Philippine Islands has succeeded in isolating a bacillus that answers to all the tests applied to the *B. coli dysentericum*. We have met with no description of its characters.

Ogata in 1892 found a short bacillus in dysenteric stools, about a quarter of the length of the tubercle bacillus, which, when introduced by mouth or rectum, caused dysenteric symptoms in animals. We have met with no further accounts of this microbe.

It seems evident from these researches—(a) that a chief place in the pathogenesis of dysentery, or of some forms of it, must be assigned to that variety of the *B. coli* communis known as the *bacterium coli dysentericum*, (b) that a virulent form of streptococcus and a small proteus, possibly also other organisms, are capable of initiating the dysenteric process, (c) that bacteria that are themselves powerless to give rise to dysentery are nevertheless active agents of mischief, some of them by increasing the virulence of the specific microbes, others by promoting suppuration and ulceration. The types of dysentery caused by the individual organisms, or their grouping, have not been determined.



**Amoebic Dysentery.**—Amoebæ are met with in the stools of healthy persons and in those suffering from cholera, enteric fever, and other inflammatory and ulcerative diseases of the bowels. A simple irritation of the intestinal canal seems sufficient to lead to their multiplication. Schuberg, for example, found amoebæ in the loose stools of ten out of twenty healthy persons to whom he had administered a purgative dose of Karlsbad salts. The frequency with which these organisms are present in the intestinal canal in health differs greatly in different regions and localities. In some parts of Italy, Greece, and Egypt, amoebæ are common parasites of healthy persons. Gasser, in Algeria, examined the stools of twenty persons in perfect health and found amoebæ in four of them. Amoebæ are more common in warm than in colder climates. This harmless *amoeba coli* is morphologically indistinguishable from the amoeba met with in dysentery, which is known as the *A. dysenteriae*. Other species of amoebæ, smaller and less readily recognised, such as the *A. guttula*, *oblonga*, *spinosa*, *diaphana*, *verruculosa*, and *reticulata*, are also frequently present in the stools of persons in health and of those suffering from various intestinal complaints, including dysentery. Their pathological effect, if any, are unknown.

The *A. dysenteriae* is a unicellular, excentrically nucleated organism, consisting of a granular ectoplasm and a homogeneous, pale green ectoplasm. It varies in diameter from 6 to 36  $\mu$ , and often contains one or more non-contractile vacuoles. It is extremely motile and locomotive, but becomes motionless at a temperature below 75° F. According to the observations of Grassi and Calandruccio, it multiplies by simple fission in liquid faeces, but when the stools are pulqueous the amoebæ become encysted. In this state they contain one or more nuclei which develop into amoebæ when ingested. Free amoebæ have been found in water and soil polluted with dysenteric evacuations, and it is probably by drinking water or contaminated food that they gain admission into the intestinal canal. These amoebæ frequently contain red blood corpuscles and bacteria. Hence it is contended by some that by engulfing and digesting the bacteria of dysentery the amoebæ are to be considered as useful auxiliaries to the phagocytic cells of the intestine. Others look upon them as destructive agents, producing softening, ulceration, and sloughing of the tissues of the bowel, and as vehicles for transporting bacteria from the bowel to the liver.

They are found in the blood-stained mucus, in the minute gelatinous masses of necrotic tissue derived from the ulcers, and in smaller numbers in the liquid stools of amoebic dysentery. They are also found in the intestinal ulcers and the surrounding zone of diseased tissue.

The numerous experiments of Kartulis, of Kruse and Pasquale, and others, prove beyond doubt that when dysenteric stools, or pus from a liver abscess containing amoebæ, are injected into the rectum of the cat, the amoebæ multiply rapidly and induce a hemorrhagic and ulcerative inflammation of the bowel. Kruse and Pasquale succeeded in producing dysenteric symptoms by the pus of a liver abscess containing amoebæ, but which was sterile as regards bacteria. These experiments, apparently so conclusive, have lost much of their value since it has been shown by Casagrandi and Barbagallo that similar symptoms and lesions follow the injection of hepatic pus containing neither amoebæ nor bacteria. In these cases the intestinal lesions must result either from the irritant nature of the injected material, or from the presence of bacterial toxins in the sterile pus. Indeed, Zancarel has repeatedly produced, not a dysentery only, but a dysentery complicated with liver abscess, containing streptococci, by the injection of hepatic pus destitute of amoebæ and sterile to culture. In these cases it is evident that the pus was either not really sterile, or that the irritation set up in the bowel by the injection had rendered virulent the harmless streptococci present in the canal, and that these had given rise to the dysentery and the liver abscess.

The experiments hitherto made in order to determine the part played by amoebæ in the dysenteric process are altogether inconclusive. It will only be when pure cultures of amoebæ have been obtained from healthy and dysenteric stools, and the experiments repeated with these, that we may hope for unambiguous results. Most of the experiments have been made on the cat, an animal in which non-specific irritants readily set up catarrhal and ulcerative inflammation of the large intestine. It has been too readily assumed in these researches that the inflammation set up in the various experiments has been dysenteric. It is still an open question whether the *A. dysenteriae* differs specifically, or at all, from the *A. coli*, and whether either can give rise to dysentery in the healthy bowel. It must also be borne in mind that in amoebic dysentery we always meet with the bacteria of ordinary dysentery, especially streptococci and varieties of the bacillus coli communis. It is, in fact, like other forms of dysentery, a mixed infection. It is possible, although this has not been proved, that the amoebæ really destroy the bacteria of ordinary dysentery, and thus convert what might have been an acute into a chronic process. But this would not in every case be a gain, for we can readily believe that the invasion of intestinal ulcers by amoebæ will have the effect of converting a disease that would otherwise have yielded to treatment into a chronic and intractable malady. Our recognition of amoebic dysentery as a distinct form rests on grounds which

are unaffected by the experiments referred to. Clinical observations associate a peculiar type of dysentery with amœbæ in the stools. Their presence in the swollen submucous tissue, in the ulcers themselves, in the spreading zone, and in the lymph spaces, proves that they are active agents in the dysenteric process.

**ETIOLOGY.—General Conditions.**—Geographical Distribution.—Non-amœbic dysentery is a ubiquitous disease in the sense that under certain conditions, such as war or famine, it may appear in any climate. As a result of famine, very severe epidemics have at different times broken out in Ireland. Up to the seventeenth century dysentery was endemic throughout the whole of Northern and Western Europe, and it occupied by no means an unimportant place in the pathology of the British Islands. It is still to some extent endemic in Sweden, especially in the neighbourhood of the central lakes, and it has also appeared repeatedly during this century in an epidemic form not only in the lake region but also in elevated and dry districts. Both in the northern and southern hemispheres dysentery increases in frequency as we approach the equator, but not by any means in a uniform way. It is notably a disease of tropical and sub-tropical countries, in many of which it takes the first place as a cause of death. Its incidence on different regions varies greatly. It is comparatively mild and rare in Singapore, in the Malayan Peninsula generally, and in British Guiana, all within a few degrees of the equator, while countries at comparatively high latitudes, such as the North-West Provinces of India, Arabia, the Mediterranean shores of Africa, Senegal, and the coasts of Chili so far south as the 33rd degree, suffer severely. The geographical distribution of amœbic dysentery is still imperfectly known. As a sporadic disease it occurs in the central and northern regions of Europe, and in the northern states of the Union. It is more common in the south of Europe and the southern states of the Union. It is known to be exceedingly prevalent in Egypt, and is probably far from rare in the tropics generally, although there is no evidence that it is the prevailing form of dysentery in warm climates. The name of "tropical" dysentery applied to this form is misleading.

**Altitude.**—The coast lands and inland valleys of tropical countries are, as a rule, more subject to dysentery than the higher lands of the interior. In India the ratio of admissions from dysentery at stations less than 100 feet above sea-level is 41.9, under 500 feet, 32.5, from 3500 to 8000 feet, 18.7, and above 8000 feet, 3.8 per 1000. But no altitude affords security from the disease unless it is sufficient to reduce the temperature to that of temperate latitudes. Moderate elevations are sometimes even more dangerous than the sea-level. Béranger-Féraud relates that in 1840 the troops in Martinique

were removed to a camp situated at a height of 1200 metres in order to escape from yellow fever, but it was found that dysentery at that elevation was as fatal as the yellow fever of the plains, and the camp had to be abandoned. The physical character of the soil, the water, and circumstances of a climatic kind—winds and atmospheric humidity—are factors which modify the influence of altitude.

**Season.**—In temperate climates dysentery is notably a disease of summer and autumn. Of 446 epidemics tabulated by Hirsch, 415 broke out from June to September. From August to October is the season of dysentery in all temperate climates in the northern hemisphere. In tropical and semi-tropical countries the incidence of the disease in summer and autumn is by no means so constant as is generally represented. In Bombay the maximum of admissions into the European Hospital falls on the coldest months. The percentage of deaths in the native army of Bengal in the three coldest months—November to January—is 39.4, in the three warmest months—May to July—16.6. It is the same in Mauritius, the dysentery season there is from May to August, months of falling temperature, and diminishing rainfall and humidity. August, the coldest month of the year, is that charged with the maximum dysentery mortality. As an epidemic disease attendant on war or famine, dysentery has often raged with great severity in winter when the temperature has been extremely low, as was the case in the Crimea in 1854-55.

Amœbic dysentery is said to be contracted in most instances in the warm season.

**Temperature.**—From what has already been said of the latitudinal, altitudinal, and seasonal relations of dysentery it may be inferred that a high temperature favours its prevalence. This view is supported by the fact that in temperate climates the years in which dysentery is most prevalent are, as a rule, exceptionally warm years. The same has also been observed to hold good in Algiers, Senegal, the West Indies, and Brazil. All experience points to sudden fluctuations of temperature in warm climates as a powerful exciting cause of dysentery. Chilly nights succeeding warm days, exposure to cold and wet after the body has been overheated, determine dysentery in countries where the disease is endemic. It may be remarked that when the temperature throughout the year is equable, the cases of dysentery are pretty evenly distributed over the whole year, but in countries where the amplitude of the annual range is great, the bulk of the cases tend to be concentrated on a few months. In temperate climates these months are summer and autumn, in tropical countries, winter, especially if the nocturnal variations during that season are also high.

**Soil.**—The geological formation of the soil

appears to have no influence on dysentery prevalence. The same cannot be said of the physical characters of the soil. We have already noticed its incidence as an endemic disease in the lake districts of Sweden. The marshy provinces of Holland recently furnished a dysentery death-rate double that of the country generally. According to Kelsch and Kienor, dysentery in France shows a predilection for marshy and moist soils. "It is thus," they say, "that the reports of the Academy continually notice its occurrence in various departments of Brittany, in the fluvial districts of the lower Loire and its affluents, in the basin of the Somme, on the plateaux of the Doubs and the Vosges. The southern part of Finistère, l'Ille-et-Vilaine, some districts of the Côtes du Nord, and above all Morbihan, have acquired in this respect a sad notoriety." To the same order of facts belong the numerous instances of outbreaks of dysentery caused by the drying up of lakes and ponds, and of deposits resulting from inundations, and cleaning out of canals and reservoirs, and the exposure of the mud to the action of the sun. The same conditions doubtless favour outbreaks of dysentery in the tropics. What are the infective agents giving rise to the disease in these instances, and in what way does infection take place? These questions do not, as yet, admit of answer.

*Relation to Malaria* — Dysentery and malaria are perfectly distinct diseases, the former may be very severe in regions where malaria is unknown, yet in the tropics they are often endemic in the same localities.

*Fæcal Pollution of Soil and Water* — These are undoubtedly among the most important factors in the etiology of dysentery. Every epidemic of dysentery is a proof of the infectiousness of dysenteric evacuations, and the disease in most of these cases seems to be spread directly or indirectly by soil and water pollution. Creighton relates an instance illustrating the way in which epidemic dysentery becomes diffused. The brig *Sandwith* with Irish emigrants suffering from famine dysentery put in at Penzance on the 7th of June 1848. Three of the women passengers died on shore of the disease. On the 16th of July the disease appeared for the first time among the natives of the town. No fewer than 500 cases and 82 deaths occurred in the town. New foci were also set up in the country districts by domestics, who, having contracted the disease in Penzance, had returned to their homes in the country for treatment. But dysentery often arises in connection with fæcal pollution of soil and water when there is no evidence of specific contamination. It is enough for an army to encamp long enough on a spot for the soil to become polluted in order to ensure an outbreak of dysentery. The epidemic of dysentery in the Cumberland and Westmorland Asylum recorded by Clouston was ascribed to

the emanations from sewage applied to fields situated at a distance of 300 yards from the ward where the disease broke out. Dysentery prevailed in the Wakefield Asylum in 1827-28. The whole sewage of the Asylum, we are told, was collected in cesspools within a few feet of the wards. In these cases the air appears to have been the vehicle of infection.

Fæcally polluted water was the cause of the numerous fatal outbreaks of diarrhoea and dysentery in the Millbank Penitentiary during the first half of this century. The water-supply was derived from the Thames as it ebbed and flowed beneath its walls. No more outbreaks occurred after a pure supply was provided. In the same way dysentery frequently occurred among the troops at Cork when their water-supply was derived from the sewage-polluted water of the Lee, and disappeared when another supply was obtained. Examples of this kind, which might be multiplied to any extent, place it beyond doubt that fæcally polluted water is capable of giving rise to dysentery. There is a good deal of evidence to show that water containing decomposing organic matter, purgative salts, and other irritating constituents, favour the outbreak of dysentery. More conclusive evidence of the part played by impure water in the causation of dysentery could not be wished than that supplied by Copinger relating to the Royal Navy. Dysentery, he says, has diminished in frequency as sanitation in respect to food and water has improved. "The proportion of cases occurring in the years 1860, 1870, and 1880 respectively, were 12.7, 3.5, and 1.2 per 1000 of all the men employed, and when we remember that the use of distilled water on board ship was coming into general use about the year 1870, the above figures are strongly suggestive of an intimate causative relation between polluted water and dysentery." Many a death from dysentery in tropical countries would be prevented if travellers and others would adopt the precaution of using only boiled water.

Tainted food, indigestible substances, unripe and over-ripe fruit, excesses of all kinds, especially in alcohol, predispose to dysentery.

*Dysentery of War and Famine* — Severe and long-continued famine is uniformly followed by dysentery, whether in warm or cold climates, but in some regions more severely than in others. Dysentery became epidemic in the kingdom of Naples during the famine of 1763. Dysentery and diarrhoea were the most fatal diseases engendered by the Irish famine of 1847-49. From Ireland the infection was carried by emigrants to the United States, where it raged from 1847 to 1856. In the recent famine in Russia dysentery was widely prevalent. Dysentery along with diarrhoea never fails to claim the largest tribute of mortality in India in famine years. In 1897, a year of extraordinary

misery in the Central Provinces, the death-rate from dysentery and diarrhoea was more than four times that in ordinary years. The same connection between famine and dysentery in warm climates has been noticed in Senegambia, Algiers, and Tunis (Hirsch). Dysentery of a spreading kind is a no less constant attendant on war, affecting alike the troops in the field and those subjected to siege. In the latter case the troops suffer in common with the civilian population. In war dysentery three sets of factors come into operation. (a) conditions which predispose the system to the disease, among which are bodily exhaustion, exposure to heat and cold, lying on damp ground, overcrowding in the case of besieged garrisons, and often scarcity and bad quality of food, (b) conditions which more directly determine infection, such as the pollution of soil and water by faecal matters, (c) facilities for the spread of the infection when the disease has once made its appearance among a densely massed body of men.

**Personal Conditions.—Race**—It is frequently stated that Europeans in warm climates are more liable to dysentery than the natives. This is not the case in India. The admission-rate per 1000 of the European army of India (1897) was 45.7, that of the native army 66.4. All ages suffer from dysentery. If we include under dysentery the hemorrhagic catarrhal complaints of infancy and childhood, dysentery makes most victims in those under five years of age. In 1878, the dysentery death-rate of the army of India was 1.73 per 1000, that of European children 3.84 per 1000. The sexes suffer in nearly the same proportion. The poor suffer more than the rich. Dysentery is more prevalent in small towns and villages than in large cities. There is no acclimatisation for dysentery. During his first year in a tropical country the European is more liable to the disease than in the two or three following years, but after the fourth or fifth year the liability appears, upon the whole, to increase, according to the length of residence in the tropics.

**PATHOLOGICAL ANATOMY.—Non-anæmic Form**—From the stand-point of pathological anatomy non-anæmic dysentery presents three varieties: (a) That in which there is no croupous or diphtheritic deposits, the lesions being inflammatory, ulcerative, and gangrenous. This, for the sake of distinction, we shall speak of as inflammatory dysentery; (b) In another variety the surface of the mucosa presents a croupous deposit, the sub-mucosa being little or not at all affected. This is croupous dysentery; (c) In a third class of cases the mucous and sub-mucous coats are the seat of exudation—diphtheritic dysentery. The croupous and diphtheritic varieties appear to be grades of the same process. The croupous form necessarily terminates in ulceration, which is principally superficial, the diphtheritic ends

in ulceration and sloughing of the mucosa and sub-mucosa, and often enough of the muscular coat. The lesions of two, or all, of these varieties may be met with in a single case.

As regards site, dysentery is generally most marked at one or other extremity of the large intestine,—the cæcum and ascending colon, or the descending colon, sigmoid flexure, and rectum. I have often seen, however, the whole of the large intestine so involved that it was difficult to say what particular part was most affected.

A catarrhal inflammation is the initial stage of all the varieties mentioned. The mucous membrane at the seat of the disease is more or less thickened, of a dark red colour, studded with ecchymotic points, and covered with blood-stained mucus. The congestion is either tolerably uniform over a large surface, or is disposed in streaks or patches affecting chiefly the mucous folds. This stage is often observed in certain parts of the bowel when the disease has already gone on to ulceration and sloughing in other places. In a case observed by Houston, in which death occurred after two days' illness, the mucous membrane of the cæcum was found to be reddened and thickened in small patches, which ran into one another like the eruption of the skin in measles. The transverse colon was mottled and thickened, the descending colon and rectum was less affected. In the rectum the mottling was mixed with red points the size of a pin's head. There was no ulceration, although the stools contained blood, and no membranous deposit had yet appeared, although the epidemic in which the case occurred was of the croupous kind. In the catarrhal stage the capillaries and small veins are engorged, the follicles are often surrounded by a zone of congestion. The epithelium is partially detached, and the solitary glands are often more or less enlarged.

When the disease in the inflammatory variety has advanced beyond the catarrhal stage small necrotic spots situated in the mucosa are often observed, along with ulceration, and it is not improbable that the ulcers in most cases start from these necrosed foci. The ulcers increase in size and depth, and are generally transverse to the axis of the gut. In severe cases, side by side with ulcers, are sloughs of an ashy or dark colour, or the affected portions may be found converted into a dark putrescent mass. Exceptionally, the whole of the large intestine is gangrenous, and on opening the abdomen the bowel may be seen, as Chevers expressed it, coiled up like a dead snake, a flaccid gangrenous mass. A case is recorded and figured by Cayley in which the whole of the mucous membrane from the cæcum to the anus presented the appearance of a suppurating sore. Whether mild or severe, no croupous or diphtheritic deposit is observed in the inflammatory form. The calibre of the bowel is not narrowed, and what-

ever may be the degree of thickening resulting from oedema or suppuration, the bowel is never rigid.

The *croupous variety*, as we have already seen, begins with congestion, ecchymosis, and thickening, followed by a croupous deposit. This presents itself either as a soft, jelly-like layer, which may be rubbed off from the mucosa, not, however, detaching the epithelium, or it occurs as a firm and adherent deposit of a grey, rusty, or black colour. In some cases it appears in isolated specks limited to the folds of the mucosa, in others it forms watery masses, discrete or confluent, involving larger or smaller surfaces of the mucous membrane. That this is not a simple necrosis of the surface of the mucosa, such as is seen in the inflammatory variety, is proved by the epithelium being found *in situ* under the adhering layer. This deposit consists of a granular, sometimes indistinctly fibrillated, substance enclosing red corpuscles and bacteria. The underlying mucosa is thickened from congestion, oedema, and increase of lymphoid elements. The surface deposit dips down into the tubular glands, which become distended by hyperplasia of their lining epithelium, compressed and distorted by pressure, sacculated by obstruction from within or by pressure from without. The solitary glands are often enlarged and prominent, and at a later period become necrosed, leaving small, round, punched-out ulcers. As the disease progresses, the deposit is detached, leaving an ulcerated surface. The ulcer is at first limited to the mucosa, but it may afterwards gain in depth and successively involve the sub-mucous and muscular coats.

In the *diphtheritic variety* the bowel becomes thickened and rigid, and its calibre narrowed. Internally, the affected portions are variegated in colour and uneven on the surface. The wall of the bowel cuts like brawn, the section presenting a streaky yellowish and red appearance. Microscopically the most notable appearances are engorgement of the blood-vessels, dilatation of the lymphatics, thickening of the walls of the smaller veins, increase of small cells, and the presence of an amorphous granular substance in the meshes of the connective tissue, the cells of which undergo degenerative change.

The necessary termination of the diphtheritic form is sloughing and gangrene. The sloughing arises in some cases from the compression of the vessels and tissues by the exudation and the multiplication of lymphoid cells, but, in other instances, arrest of the circulation of blood and lymph from the action of the virus appears to give rise to necrosis, sloughing, and gangrene.

*Amœbic Dysentery*.—In amœbic dysentery, to use the words of Laffeur, "thickening of the bowel is a constant and very characteristic feature. It may involve all the tunics, but is especially marked in the sub-mucosa, and is sometimes limited to it. It consists in a general

oedema, and in localised areas of thickening, which appear on the surface of the mucous membrane, especially its folds, as sharply circumscribed hemispherical or ovoid projections, over which the membrane is slightly reddened or discoloured. When incised they are found to contain a pale or greyish-yellow, viscid material consisting of detritus of tissues, red corpuscles, and amœbæ." When the mucous membrane gives way, ulcers are formed, which tend to burrow beneath the mucous coat, and often communicate with neighbouring ulcers. The ulcer is primarily seated in the sub-mucosa, and spreads by further infiltration and softening of the surrounding thickened sub-mucous tissue. Its edges are ragged, the base sloughy, clean, or granulating according to the stage in which it is found.

The microscopic appearances in amœbic dysentery differ little from those met with in other forms. We have the same engorgement of blood and lymph vessels, the same artematous thickening, the same increase of small cells. The connective tissue cells undergo degeneration, and the intercellular substance becomes converted into an amorphous, granular mass. The glandular structures are only secondarily affected.

The lesions of *chronic dysentery* are multifarious. As results of the primary acute attack, we meet with fibrous bands or *plaques* formed during the process of repair. These sometimes give rise to constrictions of the gut. Side by side with these may be found portions of the bowel which have undergone atrophy, involving more or less all the coats, or confined to the glandular structures. As a result of the chronic inflammatory process, we meet with more or less diffuse thickening and induration. In some cases the affected portions of the bowel are semi-cartilaginous to the feel. Chronic ulcers are also frequently met with, especially above constrictions or contractions. When death has followed an acute exacerbation the lesions characteristic of the acute form will also be present.

*ASSOCIATED PATHOLOGY*.—The mesenteric glands in acute dysentery are enlarged and hyperæmic, in the chronic disease, enlarged, pigmented, indurated, and sometimes the seat of cheesy deposits. When ulceration has ended in perforation diffuse peritonitis will be found. Much more frequently perforation is prevented by partial peritonitis, with effusion of lymph and the formation of adhesions to the neighbouring viscera. The colon may thus be adherent to the coils of the small intestine, to the stomach, to the under surface of the liver, or to the spleen; these adhesions often seriously affect the functions of the bowel. In a considerable proportion of cases of tropical dysentery, especially of the amœbic kind, the liver is the seat of solitary or multiple abscesses, or of pyemic deposits. It may also be found congested and hypertrophied

without abscess. The spleen is usually normal, never enlarged unless as a result of a malarial complication; in exceptional cases it contains suppurating foci. The kidneys in acute cases are generally healthy, but when septicæmia has developed they may exhibit the lesions of acute nephritis. In chronic cases they not infrequently present the characters of one or other of the forms of chronic Bright's disease.

**Repair**—The process of repair in all forms of intestinal ulceration is the same, and differs in no respect from what is observed in ulcers elsewhere. Granulation tissue springs up in the base and sides of the ulcer. Its oedematous or indurated edges become levelled down by absorption, overhanging portions of the mucous coat become attached to the subjacent tissue. When the loss of substance has been comparatively limited no cicatrix remains, and little beyond pigmentation marks the site of the ulcer. Extensive ulcers heal by the formation of cicatricial tissue, which restores the continuity of the bowel, but too often leads to constrictions.

**SYMPTOMS**—*Non-Anæmic Dysentery*—When the disease is epidemic, dysentery frequently sets in suddenly, the patient having up to the time of attack been in perfect health. In the endemic dysentery of warm climates cases are now and then met with in which the dysenteric symptoms, as Annesley remarks, are present "from the first hour at which the patient complains." It is in these cases that the disease is apt to be ushered in by a chill or rigor. More frequently the advent of the disease is less abrupt. The patient has been troubled for a week or more with constipation, or alternations of constipation and bilious, mucous, or serous diarrhoea. In other cases symptoms of indigestion precede the attack, which then begins as a simple diarrhoea. In whatsoever way the disease begins the first motions are usually loose and feculent, they then become mixed with blood-stained mucus. After a time the feces disappear from the stools, which now consist of little else than blood and mucus. The motions are preceded by severe colicky or griping pains and are passed with straining. Straining is most marked when the disease is seated in the sigmoid flexure and rectum. The number of motions varies according to the severity of the disease. In mild cases the stools do not exceed ten or fifteen in the twenty-four hours; in severe cases there may be as many in an hour, and the desire to go to stool is constant. Long, painful straining results in the passing of a small quantity of a gelatinous, semi-transparent, sometimes bile-coloured mucus, tinged with blood, which affords little or no relief. The patient is with difficulty induced to quit the commode. In some cases blood is more abundant, and small clots may be passed.

The constitutional symptoms during this stage are comparatively slight, even when the motions

are frequent and the pain distressing. There is little or no rise in the temperature. The pulse is slightly increased in frequency, the tongue is coated, the appetite impaired, occasionally there is nausea, but seldom vomiting unless the liver is involved. The patient is restless and irritable.

Such are the leading symptoms of the catarrhal stage. If the disease is arrested at this point the bowel suffers little damage, and recovery is usually rapid and complete if ordinary care is taken during convalescence.

The first sign of improvement is the reappearance of feces in the stools. This is followed by a decrease in the number of motions, the disappearance of the abnormal discharges, and the relief of the griping and straining.

Should the disease continue to make progress the stools undergo a change. They lose their mucous, slimy character and become watery, of a dark-red colour, not unlike the washings of meat. Their odour is distinctive, and *sui generis*, different alike from the mawkish smell of the first and the distinctly gangrenous odour of the third stage. When allowed to rest a sediment subsides, consisting of epithelium, blood corpuscles, pus cells, debris of tissue, and small shreddy sloughs. The fluid portion is found to be rich in albumen. It has been estimated that in a dysentery of moderate severity some fifty or sixty grammes of albumen is discharged daily. In this stage prolapsus of the bowel is often troublesome, and when the disease is seated in the rectum the bladder often becomes affected, and dysuria, strangury, and retraction of the testicles supervene, giving rise to more distress than that caused by the symptoms directly referable to the bowel. The constitutional symptoms during this stage are more marked. There may be more or less fever of a remittent or intermittent type, with evening exacerbations. The evening rise in the temperature seldom, however, exceeds two or three degrees Fahrenheit, if the case is uncomplicated. The pulse is fast, weak, and soft, the tongue tends to become dry, there is generally entire loss of appetite, there is thirst, nausea, perhaps occasional vomiting, the urine is scanty and may contain traces of albumen, and there is rapidly increasing emaciation and debility. The intermittent colicky pains of the first stage continue, and there is sometimes added a persistent feeling of uneasiness or distinct pain in the tract of the large intestine, increased on pressure.

If the disease now takes a favourable turn the stools become feculent and all the symptoms moderate, but the convalescence is prolonged, and is apt to be interrupted by the slightest imprudence in diet or regimen.

The transition to the third stage is marked by the stools assuming a distinctly gangrenous odour, becoming less watery, of a dark-brown coffee colour, sometimes with a greenish or

yellowish scum, and containing shreds and sloughs, and sometimes blood-clots. The sloughs are of various colour, size, and thickness. Some are thin and shreddy, of an ashy, olive, or dark colour indicating gangrene of the mucosa, others are thick and pus-infiltrated, derived from the mucous and sub-mucous coats. In comparatively rare instances tubular sloughs are discharged. I have seen sloughs of this kind several inches in length. These may project for a time from the semi-paralysed anus, causing much pain. Dutrouleau relates a case in which a patient recovered after passing nearly 14 inches of the mucous and sub-mucous coats. Another case of recovery is recorded by Fayrer in which a dark-grey tubular slough, about a foot in length, was first discharged, and then another three inches in length. It is not, however, to be concluded from such instances that recovery is anything but a very rare event when tubular sloughs consisting of the coats of the bowel, and not of croupous deposits, are discharged. The few cases of this kind that I have seen proved fatal.

When the gangrene is progressing to a fatal termination the pains subside. The anus becomes relaxed and patent, the motions pass involuntarily, the temperature often falls below the normal, the features become shrunken, hiccup supervenes, and the patient dies in a state of collapse. Typhoid symptoms, on the other hand, may develop, and the temperature then continues above the normal. Occasionally perforation occurs, and the patient succumbs to acute peritonitis. When the strength is maintained, and algid and typhoid symptoms are absent, hope of recovery is not to be abandoned.

I described in 1893 a form of dysentery in which the cecum and ascending colon are chiefly affected, which often follows or is complicated with malaria. This was the prevailing type of the disease in the Ashantee campaign of 1874. In this form the motions at first are loose, frothy, yellowish or greenish, mixed with mucus and blood, and passed with little straining. As the disease advances they become chocolate-coloured and deposit a grumous sediment of pus, blood, shreds, and sloughs. What appears to be the same type of dysentery has since been described by Babes, who supposed it to be peculiar to Roumania. Marchesava has met with similar cases in Italy, and has found it to be a mixed infection of amœbæ, bacillus coli, streptococci, staphylococci, and other bacteria. This disease runs an acute course and seems to be only met with in malarious regions.

We have tried to depict the more common features of non-amœbic dysentery, but every individual case presents certain peculiarities, and considerable differences in respect to particular symptoms are sometimes observed. In some cases, for example, the stools remain feculent throughout the catarrhal stage, or there

is an occasional motion of green or yellow feculent matter. The frequency of the stools is not always in proportion to the severity of the disease. I have notes of fatal cases in which the stools have not exceeded twelve in the twenty-four hours. Sloughing occasionally takes place when the mucous stools would indicate that the disease is in the first stage. The second and third stages are occasionally not defined. Vomiting is sometimes an urgent symptom when, after death, no disease of the liver is found. It may finally be noted, as a point of some prognostic importance, that hiccup may persist for days in comparatively mild cases. The gravity of this symptom when combined with prostration and a typhoid condition is well known.

*Amœbic Dysentery* is gradual, as a rule, in its onset, intermittent in its progress, and protracted in its course. It generally runs on for many months, and sometimes for one or two years. It begins for the most part with a painless diarrhoea, with intervals in which the motions are formed or the bowels constipated. The stools during the exacerbations are loose and yellowish, and contain mucus, and occasionally a little blood. This state of things may continue pretty much throughout the tedious course of the illness, the patient losing in flesh, and becoming weak and anæmic. The real nature of the malady may only be discovered when symptoms of liver abscess supervene. In more severe cases the periods of quiescence are short, the exacerbations frequent, protracted, and severe. During the exacerbations the stools are loose or watery, yellow, greenish, or grey in colour, and contain mucus, with streaks or clots of blood, and small gelatinous necrotic masses derived from the intestinal ulcers. The patient suffers from colicky pains, but there is little tenesmus.

Grave symptoms may appear suddenly in a case that had previously been running a mild course, or the successive exacerbations may increase in severity until the disease assumes a dangerous type. In this grave form the patient suffers from severe colicky pains, and in some cases from a minor degree of tenesmus. The motions number twenty or thirty daily. At first they are scanty and consist chiefly of blood and mucus, at a later period they become more copious and watery, are extremely offensive, and contain debris of tissue and sloughs. When these cases run on to a fatal termination the general symptoms are those of the gangrenous dysentery already described.

This severe form may be primary, in which case the disease begins abruptly and runs on in a few weeks to gangrene. It will probably be found that in these acute cases the bacteria of ordinary dysentery play the principal rôle.

*Chronic Dysentery*—Chronic dysentery either begins insidiously, developing out of a simple diarrhoea or a subacute form of dysentery, or it

is a sequel to the acute disease. Many of the cases of tropical dysentery which begin insidiously as an intermittent diarrhoea are probably amoebic, but we meet with cases, chronic from the beginning, which are clearly non-amoebic. These cases often run a much more protracted course than amoebic dysentery. We meet with instances of this variety which persist with intervals of quiescence for seven, ten, or fifteen years.

Chronic dysentery is, however, generally a sequel of the acute disease. The acute symptoms subside, but a tendency to looseness persists, with occasional traces of blood and mucus in the stools, and recurring attacks of colicky pains or of abdominal discomfort. These symptoms disappear for a time, and the patient resumes his ordinary habits, when a subacute exacerbation shows that the primary attack had left behind it latent mischief which a chill or some slight indiscretion in diet sufficed to stir into activity. Now follow periods of quiescence, during which for a few days or weeks the bowels may be normal or constipated, the prevailing condition, however, being a tendency to looseness. These alternate with periods of exacerbation during which the stools are dysenteric and passed with griping and stinging. The appetite after a time is lost or becomes capricious, the digestion is impaired, often there is nausea, occasionally vomiting, and the patient emaciates and becomes anæmic.

When the bowel has become narrowed by cicatrices, or its action impeded by adhesions resulting from the primary attack, or by thickening set up by the chronic inflammation, the symptoms of intestinal stenosis develop. The evacuations are passed with difficulty, the abdomen becomes tumid and tender, there is a distressing feeling of distension, the breath acquires a feculent odour, the complexion becomes earthy, the tongue glazed, the skin dry, and the patient sinks from exhaustion or is carried off by some intercurrent malady.

**SPECIAL FORMS AND COMPLICATIONS.**—*Malaria*.—We must distinguish between dysentery complicated with malaria, and dysentery caused by malaria. When the patient is not immediately suffering from malarial paroxysms, the fact of his having recently done so sometimes shows itself either in an evening rise of temperature or in a return of aguish attacks. In many instances the previous malaria has no effect whatever on the course of the dysentery. An evening rise of from one to three degrees is often ascribed to malaria when it is really caused by septic absorption. When dysentery coexists with actual paroxysms of malarial fever, the two diseases may run their course without the one perceptibly influencing the other. In other instances the dysenteric symptoms become aggravated during the paroxysm, but the contrary result is also occasionally observed, the dysenteric symptoms subsiding during the fit.

There is a form of pernicious malarial attack which is characterised by intermittent dysenteric attacks. The symptoms subside or disappear during the intermission to recur at quotidian or tertian intervals. This is an exceedingly rare form of pernicious fever. Much more common is an intermittent intestinal hæmorrhage. The blood passed may be pure or mixed with feces. In these cases we have to do with malaria, not with dysentery. But malarial fever may give rise to perhaps more than one special form of dysentery, which may be looked upon as substantive diseases. The fever-stricken troops in the great fever epidemic in Mauritius in 1866-67 were attacked with a dysentery characterised by discharges of a thin, smoky, dark fluid, with no trace of feculent matter. Sometimes sloughs were passed, sometimes none. There was a marked tendency to collapse. On autopsy a total sloughing of the large intestine was observed in some cases, while in others the only morbid appearance was a prominent state of the glands. Ipecacuanha was of no service in this form of dysentery, but large doses of perchloride of iron were frequently useful.

*Scorbutus*.—In persons suffering from scorbutic dysentery begins as a diarrhoea, and throughout its course the motions are generally more copious and less frequent than in the ordinary forms of acute dysentery. The stools consist of feces, mucus, and a considerable amount of sanguineous fluid, often with sloughs. The tormina and tenesmus are less severe. The ordinary symptoms of scorbutus will, of course, be present. It must be remembered that if scorbutus predisposes to dysentery, so does chronic dysentery predispose to scorbutus. The combination of these two diseases forms one of the most fatal maladies in besieged cities.

*Rheumatism* of the large joints has been very common in some epidemics of dysentery in Europe. One joint after another may be attacked, or the disease may be confined to one joint, generally the knee. The swelling and pain are considerable, but it is rarely accompanied by fever, and still more rarely does it terminate in suppuration. It is analogous to the joint affection of Malta fever.

*Paraplegia* of a reflex character is an occasional sequel both of acute and chronic dysentery.

*Epidemic Gangrenous Rectitis*, the Caribi or Buck sickness of British Guiana, is a highly infectious malady, which has repeatedly occurred in destructive outbreaks among the Indian population. It has also been met with in Trinidad, Brazil, and Peru, in which places the white population has not been spared. The same, or a similar disease, combined with a gangrenous stomatitis, is said by Corney to be one of the most fatal diseases in Fiji and some of the other islands of the Pacific. The



leading symptoms are those of gangrene of the rectum with dilatation of the sphincter ani. The disease is generally limited to the rectum, but it occasionally attacks the transverse colon primarily, hence a distinction is made between the "high" and "low" forms. In other instances the disease beginning in the rectum afterwards involves the whole of the large intestine.

**DIAGNOSIS**—In *simple* and *ulcerative colitis* the stools contain blood and mucus, and in the ulcerative form, pus and sloughs are present, and liver abscess may develop. The chief points of distinction are that in colitis we do not have the serous stools like washings of meat, nor the same constant desire to go to stool, nor the severe tormina and tenesmus generally met with in dysentery.

*Rectitis* differs from dysentery in this, that although there are frequent stools of a dysenteric kind, with tenesmus, the patient passes healthy motions daily or every other day, according to his habit.

*Bilharzia* disease of the sigmoid flexure and rectum simulates subacute and chronic dysentery. An examination of the stools, and of the mucosities detached from the *Bilharzia* infarctions of the bowel, for the eggs of the parasite, will reveal the true nature of the disease.

The diagnosis of the amoebic form will depend on the discovery of the amoebae in the mucus, the gelatinous necrotic masses, and the faeces. The stools should be kept at the temperature of the blood from the time they are passed until the examination is finished.

The diagnosis of the accidents arising during dysentery seldom present much difficulty. *Invagination of the bowel* occasionally occurs during the course of dysentery and diarrhoea in children. The sudden change in the condition of the patient, the supervention of vomiting, at first of the contents of the stomach, and finally of feculent matter, the tympanitic state of the abdomen, pain on pressure at the site of the invagination, without generalised tenderness, indicate the nature of the accident. The symptoms of *perforation* are those of acute generalised peritonitis, and cannot be overlooked or mistaken. *Acute limited peritonitis* resulting from inflammation of the serous coat of the bowel, consequent on an ulcer penetrating to or below the muscular coat, gives rise in many cases to more or less generalised abdominal pain, but the tenderness is limited to the part in which the disease is seated. The abdomen is not tympanitic, and the constitutional symptoms of perforation or invagination are absent.

**MORTALITY AND PROGNOSIS.**—The case mortality in the army of India, which was about 11 per cent in the ten years ending 1857, fell to 3·3 per cent in the five years 1871-75. A corresponding decrease has taken place in

the French army. In war times, not only does the number of cases increase, but also their fatality. In the German military practice during the war of 1870-71 the case mortality was 6 per cent.

The marked decrease in the case mortality of dysentery during the last half-century is in part, no doubt, the result of improved methods of treatment, but to a much larger extent it is the effect of improved hygiene, which has at once lessened the prevalence and the severity of the disease. The army returns include many cases of the mildest forms of the disease that are dysentery in little else than the name. A more just idea of the gravity of the disease is to be gathered from the mortality in cases of sufficient gravity to require admission into civil hospitals. In the Calcutta Hospital (1879) the ratio of deaths to admissions was 22·2, in Mauritius (1888) it was 22·7 per cent.

The prognosis must always be guarded, as the mildest cases are liable to undergo unexpected aggravations, and this is especially true of amoebic dysentery. In epidemic dysentery the type of the outbreak has also to be taken into account, as some epidemics are much more fatal than others. The passage of the disease from the mucous to the serous stage, notwithstanding early and efficient treatment, renders the prognosis more grave. The immediate danger is increased, and the risk of the disease becoming chronic is greater. The prognostic significance of sloughs in the stools depends not only on their number, size, and thickness, but also very much upon the way in which the patient supports the disease. When the stools present a gangrenous odour and the patient is prostrate, the prognosis is grave even if there are no sloughs in the stools. Typhoid symptoms, or a tendency to collapse, are ominous, and all the more so if hiccup is conjoined with these symptoms.

In forming a judgment respecting the probable issue of a case of chronic dysentery we have to take into consideration not only the severity of the symptoms, the duration of the disease, its effect on the patient's health, the presence or absence of contractions and adhesions interfering with the functions of the bowel, but we must also take into account the ability of the patient to obtain change of climate, rest, and care, and his readiness to submit to those restrictions in respect to diet and modes of life upon which the success of treatment largely depends.

**PROPHYLAXIS**—The methods now being tried of producing an immunity by the use of prophylactic serum are as yet outside the sphere of practical medicine, and our chief hope will probably always lie in obviating the predisposing and exciting causes of the disease. The recognition of the infectious nature of dysentery lies at the root of its prophylaxis. Dysenteric stools should be disinfected, and either deeply buried

at a distance from dwellings or excrement. Soil in the neighbourhood of dwellings and drinking-water should be guarded from faecal pollution. In countries where the disease is endemic the precaution of boiling the water before using it, unless it is above all suspicion, should never be neglected. The influence of muscular exhaustion, of exposure to cold after being heated, of insufficient nourishment, of excesses in food and alcohol, in predisposing to dysentery must be borne in mind. Other measures of prevention will be deduced from a careful study of the etiology of the disease.

**TREATMENT.**—Of primary importance in the treatment of all forms of dysentery are rest and diet. The patient is to be strictly confined to bed throughout an acute attack, whether mild or severe. Even motion in bed is to be avoided as far as possible. The bed-pan should be used to obviate the necessity of the patient getting up to stool.

Fresh milk given lukewarm is, perhaps, the best diet in most cases. When fresh milk cannot be obtained, condensed milk may be substituted. When ordinary milk is not well digested, peptonised milk, or milk diluted with lime water, may be found to answer. In a considerable number of cases milk in any form disagrees. Manson remarks that when the tongue is coated milk is often not well borne. This is true, but it also occasionally disagrees with patients when the tongue is clean. In these cases good beef tea, chicken soup, or barley water may be substituted. Whatever may be the food selected, it should be given in small quantities in the intervals between the doses of ipecacuanha, if this remedy is used. As a rule, alcohol is not only unnecessary but hurtful, but in some outbreaks the free use of wine and water has done good.

The casual indication, which is to destroy the pathogenic agents of the disease, can only be imperfectly carried out. Salol has been recommended by Rasch, Fisch, and Kartulis. It may be given in 15 to 20 grain doses in cachets, and may be used along with castor oil or other purgatives. Naphthalin has been given by mouth and enema with some success by Roessbach and Novikoff. It may be given in 15-grain doses four times daily by the mouth, or in enema dissolved in olive oil or suspended in some mucilaginous menstruum.

Benzo-naphthol appears to be deserving of further trial. It has little toxic power. It passes through the stomach unchanged, breaking up in the intestine into beta-naphthol and benzoic acid. It is specially indicated when the kidney is diseased. Thirty to fifty grains or more may be given daily in divided doses. In the present state of our knowledge these remedies are rather to be looked upon as auxiliaries to other methods of cure, than as means of cure to be trusted to alone.

The indications from the disease are to prevent the healing process being disturbed by the passage of excrementitious matter over the inflamed surface, and to secure rest for the bowel by the use of sedatives, such as Dover's powder. Of the importance of a non-irritating diet enough has been said. Experience proves that there is danger in attempting to arrest the peristaltic movements of the bowels by opiates. Many cases may recover under such treatment, because many cases will recover without any treatment other than rest and dieting, or in spite of inappropriate treatment. I have seen this method of securing mechanical and physiological rest for the bowel tried on a large scale by an eminent physician, and I am bound to say that the results were disappointing.

The symptomatic indications are to relieve pain and local irritation. Small opiate enemata are occasionally useful when tenesmus and dysuria are distressing. Poultices, fomentations, and turpentine stipes often afford considerable relief. Poultices if they are to be of service should cover the whole abdomen, and be renewed *before* they begin to feel cold to the patient. A warm bath given at the commencement of the disease is useful if care be taken to prevent a chill.

Quite recently serum methods of cure have been tried, but their value has not yet been demonstrated.

The most successful treatment of dysentery is purely empirical, and consists in the use of ipecacuanha, salines, or mercurials. Ipecacuanha and salines are alternative remedies adapted to the same forms of the disease. In severe cases if there is no contra-indication to its use ipecacuanha should, I think, be employed. Salines are to be preferred in the case of young children, delicate persons, pregnant women, and when ipecacuanha cannot be tolerated. Mercury is to be resorted to in those cases only in which the other remedies have failed, and in epidemics in which they have been found inefficacious. As it is impossible to distinguish from the symptoms the class of cases likely to be benefited by one or other of these remedies, the ipecacuanha or saline treatment should always be employed in the first instance, and receive a fair trial.

**Use of Ipecacuanha.**—The method of using ipecacuanha is as follows.—The patient should abstain from food and drink for three hours. A large emesis is to be placed over the abdomen and 30 to 60 grains of ipecacuanha powder are to be administered in bolus or cachet. More or less nausea will result, and after a time vomiting generally ensues. If the drug is retained for three-quarters of an hour or longer its curative action will not be impaired even if a considerable portion of the powder is rejected. These doses are to be repeated twice or three times daily, according to the urgency

of the case and the tolerance of the remedy, and are to be persisted in until the symptoms subside or until it is evident that the treatment is ineffectual. No advantage in the way of preventing nausea is to be expected from a reduction of the dose, for large doses often give rise to less disturbance than small ones. Nor is vomiting, unless excessively protracted, to be looked upon as something to be avoided. On the contrary, by emulging the bile-ducts, by causing free perspiration, and by the general succussion of the system to which it gives rise, the emetic action of ipecacuanha is salutary in dysentery. Should the remedy be rejected shortly after it has been taken, twenty drops of laudanum should be given before the next dose, the mustard plaster being again applied.

The first sign of improvement is usually the appearance of faeces in the stools, followed by an abatement of all the symptoms. The remedy should be continued at longer intervals until the blood and mucus has disappeared from the stools. If diarrhoea continues, it may be treated with Dover's powder or bismuth.

De-emeticised ipecacuanha has been recommended in the treatment of dysentery, but its value as a substitute for the ordinary powder is doubtful.

**Saline Treatment.**—In the saline treatment the sulphate of sodium is the salt generally preferred, as being less nauseous and irritating than the magnesium sulphate. Three-quarters of an ounce, more or less, according to the age of the patient and the effect of the drug, is to be given either in a single dose in the morning, or in smaller doses repeated every half-hour until its purgative effect is manifest. These doses are to be given daily until the stools lose their dysenteric character. Given in this way its purgative action is over before night. Trousseau was of opinion that the cure is the more certain the greater the number of the evacuations. I quote this opinion to emphasise the fact that no danger is to be apprehended from free purgation, while the employment of small irritating doses is worse than useless. I conceive, however, that excessive purging is not without danger. Some recommend 60-grain doses of Epsom salts with 10 minims of dilute sulphuric acid every hour, until the bowels are freely opened, instead of sodium sulphate.

Other purgatives may sometimes be preferred to salines. Birch advises the use of castor-oil emulsion in the dysenteric affections of children, after having cleared out the bowel by a dose of the common oil. "In a couple of days the motions will lose their slimy, bloody, and curdy appearance, and the case is resolved into one of simple diarrhoea." It may be added that castor oil has been successfully used in the dysentery of adults.

**Use of Calomel.**—Calomel administered in scruple doses twice or three times in twenty-

four hours was at one time looked upon as a specific in dysentery. Trousseau and Bretonneau tried this treatment with success in an epidemic at Touraine, but had to abandon it on account of the salivation it occasioned in some subjects. When given in these doses its action is probably chiefly evacuant, like that of sulphate of sodium. Ipecacuanha or salines being safer, this method of treatment has been abandoned.

In some epidemics calomel given in moderate doses has proved serviceable when ipecacuanha and other remedies have failed. Niemeyer is of opinion that in the higher grades of dysentery one grain of calomel with a quarter of a grain of opium given every two hours is the best treatment. His experience was derived from the epidemic type of the disease in Europe. Scheube, whose authority carries great weight, gives calomel in doses of 1 to 7 grains every four or six hours, obviating constipation, if necessary, by an occasional dose of castor oil. He has not found salivation to result when so given. He adopted this treatment in preference to any other in the dysentery of Japan, where the disease is frequently epidemic and probably of the diphtheritic type. My experience of the use of calomel has been limited to cases which proved rebellious to ipecacuanha, and I have given it in grain doses combined with a quarter of a grain of opium, at hourly intervals for the first few doses, then at longer and longer intervals. When a case of dysentery has been treated with ipecacuanha in large doses from the beginning, and notwithstanding passes into the serious stage, perseverance in the use of ipecacuanha is not likely to prove of service, and I think calomel should be at once resorted to. If, however, the case has not been treated from the commencement, ipecacuanha should not be abandoned because the disease passes into the serious stage, but should be persevered with until it has had time to act. Calomel should neither be resorted to precipitately nor its use delayed until the case is hopeless. When the first cases in an epidemic prove inamenable to ipecacuanha it will not, of course, be necessary to begin every case with ipecacuanha or salines, but if calomel proves serviceable it should be given from the beginning.

I have found frequent fractional doses of calomel or grey powder of great service in the dysentery of children. In some forms of bowel complaint in children, with frequent slimy, bloody stools, a grain of the perchloride of mercury dissolved in ten ounces of water and given, as Ringer recommends, in doses of 30 to 60 minims hourly, is very useful.

When distinct paroxysms of malarial fever accompany dysentery, quinine is to be given along with ipecacuanha or salines. If dysentery symptoms intermit, the disease is to be treated by full doses of quinine alone. We have mentioned a form of dysentery associated with

malarial fever in which ipecacuanha was useless, but which yielded to perchloride of iron in large doses. This observation should be borne in mind. In the form of dysentery in which the cæcum is chiefly involved, and which is often complicated with a severe type of malarial fever, the great prostration contra-indicates the use of ipecacuanha. Quinine and small purgative doses of castor oil, with 20 to 30 minims of oil of turpentine, have seemed to me useful in this form of the disease.

When complicated with scorbutus, the patient should be put on milk diet. A free use of lime or lemon juice is indicated. Grapes, oranges, or pomegranates may be given when lemons cannot be procured. Bael fruit given in the form of sherbet is recommended by Maclean. Solution of the permanganate of iron, or oil of turpentine, 15 to 20 minims in almond emulsion, are the best means of checking the hemorrhage in this form of the disease.

Pain and swelling of the joints are to be treated by local applications of opium and belladonna, the affected joint wrapped in cotton-wool and swathed with flannel.

In severe hemorrhage arising from erosion of a large vessel, indicated by a copious discharge of clotted blood, astringent injections may be of use if the seat of the lesion is in the rectum or sigmoid flexure, otherwise our chief reliance must be placed in hypodermic injections of ergotin. The full dose of 10 minims of the pharmacopœial preparation should be employed.

In epidemic gangrenous rectitis injections of lemon juice, suggested by native methods of cure, should be tried, the bowels at the same time being acted on by gentle purgatives.

The medicines chiefly employed in the treatment of amœbic dysentery are calomel, salol, and quinine given by the mouth, and solutions of quinine, corrosive sublimate, and nitrate of silver in enemata. As an injection quinine is used of a strength of 1-1000 or 1-500, corrosive sublimate 1-5000, and nitrate of silver, 30 grains to a quart. One or two pints of these solutions are to be injected three or four times daily. The results hitherto obtained from these remedies have been somewhat disappointing.

**CHRONIC DYSENTERY.**—A patient suffering from chronic dysentery contracted in the tropics should be invalided home without delay. If the patient's return falls in winter, it is better, if his circumstances permit, that he should pass a month or two on the Riviera. In any case, great care should be taken as regards clothing. Flannel underclothing and the use of an abdominal belt should be insisted upon. The disease is one which gives rise to great anemia and debility, the diet, therefore, should be nourishing and easily digested. During the periods of quiescence meat should be allowed. Graves remarks in one of his lectures that

cases of chronic dysentery he had met with, "which had obstinately resisted the most varied remedies assiduously employed, got well after a liberal allowance of meat was given, and the first thing I should do," he says, "when called upon to treat a case of dysentery of long standing, would be to put my patient on a full meat diet." During the acute or subacute exacerbations the patient should be put upon the diet recommended for the acute disease. Milk and light farinaceous foods generally answer best during these exacerbations.

Rest in bed should only be enjoined during an exacerbation. Exercise, short of fatigue, is beneficial. When the patient is unable to walk, carriage exercise should be taken. The chances of recovery depend largely upon attention to these details.

Acute and subacute intercurrent attacks are to be treated on the general principles applicable to the acute disease. Instead of the powder of ipecacuanha, an infusion of 100 grains each of ipecacuanha root and sassafras bark in ten ounces of water, given in ounce doses three or four times a day, is to be preferred. When the acute symptoms abate and the stools become diarrhoeal the dose is to be reduced, and a few drops of laudanum added. Constipation is to be obviated by the gentlest laxatives. When, on the other hand, looseness is troublesome, Dover's powder, salicylate of bismuth, and bael fruit are the most trustworthy remedies. Injections of nitrate of silver or sulphate of copper are of value when no acute symptoms are present, especially if chronic ulcers are present in the lower part of the bowel. A wet compress to the abdomen is often useful when the bowel is congested or ulcerated. Enollient enemata and gentle purgatives will be required when catarrhical contractions impede the evacuation of the bowel.

**Dysgenesis.**—Any morbid state of the generative organs or reproductive powers, or, specially, hybridity in animals in which the offspring are sterile among themselves, but possibly fertile with individuals of the original races.

**Dysgeusia.**—Imperfect or depraved taste. See *HYSTERIA (Sensory Disorders)*, *PREGNANCY, AFFECTIONS AND COMPLICATIONS (Digestive and Sympathetic Disturbances)*.

**Dysidrosis.**—Excessive secretion by the sudoriparous glands and its effects on the skin, cheiropompholyx. See *SKIN, BACTERIOLOGY OF (Cheiropompholyx or Dysidrosis)*.

**Dyskinesia.**—Difficulty or impairment of walking. See *LABOUR, INJURIES TO THE GENERATIVE ORGANS (Pelvic Articulations)*.

**Dyskyesis.**—Morbid or complicated pregnancy, or, specially, extra-uterine gestation.

**Dyslalia.**—Difficult or indistinct speech from defects in the speech-apparatus apart from the cerebral portions of the same. *Dyslogia* is difficulty of speech due to a cerebral lesion

**Dyslexia.**—Difficulty in reading, word-blindness, dysagnosia. See *APHASIA (Word-Blindness)*

**Dysmenorrhœa.**—Painful or difficult menstruation, the pain being sufficiently severe to prevent the patient following her usual avocation, and being felt either in the back (sacralgia) or in one or other of the iliac regions, or in the hypogastrium and thighs. See *MENSTRUATION AND ITS DISORDERS (Dysmenorrhœa)*, CURETIAGE (*Indications, Dysmenorrhœa and Sterility*), GYNECOLOGY, DIAGNOSIS IN, HYDROPATHY, MITTSCHMERZ, MAMMARY GLAND, DISEASES OF (*Chronic Lobular Mastitis*), UTERUS, MALFORMATIONS, DISPLACEMENTS, INFLAMMATIONS, AND TUMOURS, etc. *Dysmenorrhœa* may be *congestive, endometritic, idiopathic, inflammatory, intermitte (Mittelschmerz), intermenstrual, membranous, nervous, neuralgic, obstructive, ovarian, rheumatic (or gouty), spasmodic, tubal, uterine, or vaginal*

**Dysmimia.**—Difficulty or inefficiency in producing signs in lieu of speech or in illustration of it

**Dysmnnesia.**—Defective memory

**Dysmorphia.**—Deformity, malformation. See *DEFORMITIES, TERATOLOGY*, etc

**Dysmyotonia.**—Muscular atony, as in dysmyotonia (or myotonia) congenita or Thomsen's disease

**Dysnusia.**—Weakness of mind or impairment of intellect

**Dysodia.**—Factor, especially fatid exhalations from the body, *eg* from the skin in dysodia cutanea (*hormidioria*)

**Dysodontiasis.**—Difficult dentition

**Dysootocla.**—(Ovarian dysmenorrhœa (Barries) or difficult ovulation

**Dysorexia.**—Diminished or depraved appetite.

**Dysosmia.**—Impaired or diminished sense of smell, or (sometimes) a fatid exhalation

**Dysostosis.**—Defective development of a bone, *eg* the *cleido-cranial dysostosis* of Mario and Santoni, in which there is absence or imperfect formation of the clavicle with associated cranial deformities (*pseudo-hydrocephalus*).

**Dyspareunia.**—Painful sexual coition, pain during coitus. See *GYNECOLOGY, DIAGNOSIS IN, VAGINA, DISORDERS (Vaginismus)*

**Dyspepsia.**—The morbid state in which digestion is accompanied by pain or accomplished with difficulty, numerous varieties have been described, such as *acid dyspepsia, acute, alkaline, atonic, bilious, bulimic, catarrhal, congestive, climacteric, diabetic, duodenal, fermentative, flatulent, functional, gaseous, gastro-intestinal, gouty, hepatic, hysterical idiopathic, inflammatory, intestinal, irritative, henteric, muscular, nervous, neuralgic, ovarian, pancreatic, reflex, renal, rheumatic, salivary, sympathetic, tabetic, tobacco, uramic, and uterine dyspepsia*. See also *INDIGESTION, ABDOMEN, CLINICAL INVESTIGATION OF (Symptoms), ALCOHOL (Indications in Diseases of Infancy and Childhood), ATROPHY, INFANTILE (Symptoms), BREATH (in Dyspepsia), BRONCHI, BRONCHITIS (Etiology, Predisposing Causes), CHOLERA NOSTRAS (Diagnosis, Fermentative Dyspepsia), GASTRO-INTESTINAL DISORDERS OF INFANCY (Chronic Vomiting), GALL-BLADDER AND BILE DUCH, DISEASES OF (Affections), HYDROPATHY (Dyspepsia), HYPNOTISM (Dyspepsia), LIVER, DISEASES OF (Cirrhosis, Symptoms), LIVER, PORTAL THROMBOSIS (Symptoms), MILK (Therapeutic, Koumiss Cure), NEPHRITIS (Renal Cirrhosis, Dyspepsia), SPINE, SURGICAL AFFECTIONS OF (Spinal Curves, Diagnosis), STOMACH AND DUODENUM, DISEASES OF (General Etiology, General Symptomatology, Special Symptomatology, etc), TEETH (Dental Caries), TETANY (Causation, Gastro-Intestinal Affections), TOXICOLOGY (Lead - Poisoning, Symptoms), UREMIA (Chronic, Symptoms, Digestive), WATER (Hard Water, Dyspepsia)*

**Dysphagia.** See also *ABDOMINAL ANEURYSM (Pressure Symptoms), AORTA, THORACIC ANEURYSM (Symptoms, Dysphagia), BRONCHI, BRONCHIAL GLANDS (Pressure-Effects), GLOSSOPHARYNGEAL NERVE (Clinical Features), HYSTERIA (Digestive Disorders, Dysphagia), LARYNX (Laryngeal Phthisis), LARYNX, MALIGNANT DISEASE OF (Symptoms), MEDIASTINUM (Growth, Symptoms), ESOPHAGUS (Growth, Symptoms), PHARYNX, RETRO-PHARYNGEAL ABSCESS (Symptoms), THYROID GLAND, MEDICAL (Goitre, Symptoms)*—Before referring to the various conditions that may induce dysphagia or difficulty of swallowing it is advisable to briefly consider the physiological processes governing the act

The act consists of three stages—the first being entirely voluntary, the second partially so, and the third entirely beyond the control of the will. The first stage consists of the passage of the food as far back as the anterior arch of the fauces, this being accomplished by the intrinsic muscles of the tongue and muscles of the cheek and mouth. In the second stage the

tongue is retracted, the glottis is closed, the larynx, soft palate, and pharynx are raised, and the bolus of food is brought into the posterior part of the pharynx. During this stage the food is prevented from passing into the nose by the combined action of the tensor and levator palati, and the larynx is closed effectively both at its upper and lower parts by the approximation of the true and false cords. The third stage is reached when the food descends through the grasp of the constrictors and enters the œsophagus—when by means of a twofold movement, the longitudinal muscular fibres being retracted and the circular fibres contracted, the food is propelled into the stomach.

The causes of dysphagia will readily be understood from a consideration of the structures involved in the process of normal deglutition. These causes may be summarised as follows—

(1.) Acute inflammatory conditions of the tonsils, pharynx, and larynx. In some cases of sore throat the dysphagia is out of proportion to the severity of the inflammation, a condition probably due to interference with the action of the superior constrictor muscle.

(2.) Involvement of these structures in the course of a general disease, as phthisis, syphilis, or malignant disease.

(3.) Inflammatory and other conditions in the neighbourhood not directly associated with these structures, e.g. parotitis, etc.

(4.) Stricture of the œsophagus, either of a functional or organic nature. The most common causes of the latter are malignant disease and aneurysmal tumours. Great care is necessary in the measures taken to diagnose this condition. Stricture also results from cicatricial contraction after the swallowing of various corrosive irritants.

(5.) Paralytic conditions, e.g. bulbar paralysis, post-diphtheritic paralysis, or the very late stages of progressive muscular atrophy. Any organic lesion which interferes with the integrity of the centre in the medulla, or of the efferent fibres passing to the muscles involved, may induce dysphagia. The difficulty in swallowing observed in many cases of general asthenia shortly before death is due to a general failure of the vital powers, and is thus of a paralytic nature. In these cases, doubtless, the failure is in great part dependent on a lesion higher than the medulla.

(6.) Functional Dysphagia.—This constitutes the most interesting group of all. It may be met with at any point of life, but is most common in young adults, and in very neurotic children of from ten to thirteen years old. Like other manifestations of hysteria it is more frequent in women. The clinical picture varies considerably in accordance with the greater or lesser development of other hysterical symptoms. The age of the patient, combined with a study of the temperament, history, and

general state of the patient, readily suffices to distinguish this condition.

**Dysphasia.**—Difficulty of speech due to cerebral lesions.

**Dysphonia.**—Imperfect or difficult or painful phonation or vocalisation, e.g. in cases of clergymen's sore-throat (when it is called *dysphonia clericorum*) or at puberty (*dysphonia puberum*), it may be accompanied by stammering (speaker's cramp or *dysphonia spastica*), and in it the voice may be reduced to a weak whisper (*dysphonia susurrans*).

**Dysphrasia.**—Defective speech due to defective intellect.

**Dysphrenia.**—Mental disorder.  
**Dyspnœa.**

See also ABDOMINAL ANEURYSM (*Pressure Symptoms*), AORTA, THORACIC ANEURYSM (*Symptoms*), APHYXIA (*Definition*), ASTHMA (*Symptoms*); BRONCHI, BRONCHIAL GLANDS (*Pressure*), BRONCHI, BRONCHITIS, CHEST, CLINICAL INVESTIGATION OF THE (*Inspection*), CHLOROSIS (*Symptoms*), DIABETES MELLITUS (*Diabetic Coma, Symptoms*), HEART, MYOCARDIUM AND ENDOCARDIUM (*Symptomatology*), HÆMIPLEGIA (*Disorders of Respiratory Organs*), LUNG, TUBERCULOSIS OF (*Symptoms*), LUNG, EMPHYSEMA OF (*Symptoms*), LUNG, VASCULAR DISORDERS (*Pulmonary Embolism*), MEDIASTINUM (*Growth, Symptoms*), PNEUMONIA (*Clinical Features*), STOMACH AND DUODENUM, DISEASES OF (*General Symptomatology*), TABES DOBRALES (*Affections of Cranial Nerves in*), THYMUS (GLAND Simple Enlargement, *Dysplasia*), THYROID GLAND, MEDICAL (*Exophthalmus Goitre*), URÆMIA (*Chronic, Symptoms*).

THE causes of dyspnœa will be readily appreciated on consideration of the neuro-muscular mechanism of respiration. As this is described in detail elsewhere, it is unnecessary to do more than indicate that during the inspiratory phase, air passes through the nose, pharynx, larynx, trachea, large and small bronchi, into the lung tissue proper, while during expiration, the air passes from the smaller bronchi through the larger bronchi, larynx, posterior and anterior nares, to the exterior.

Under normal circumstances this takes place noiselessly, is unattended with distress or difficulty of any kind, and has a frequency proportionate to the age and sex of the individual. In all cases of embarrassed respiration, the cause must be sought in some interference with the neuro-muscular mechanism of one or more parts of the respiratory tract. Hence the importance of making a systematic examination.

*Inspiratory Dyspnœa* may depend on obstruction in any part of the respiratory tract. The diagnosis of the site of obstruction is frequently readily determined by careful inspection of the

patient and by analysis of the points made out on extra auscultation. Thus, if there be any obstruction in the nose or naso-pharynx the respiration is of a noisy character, usually described as sniffling, and the voice is more or less nasal in quality. In cases of obstruction due to post-nasal growths (adenoids) the history of the case and general physiognomy of the patient will assist diagnosis. If the pharynx be the seat of obstruction the breathing is also markedly rough in character, the voice is thick, and deglutition impaired. When the seat of obstruction is in the larynx the quality of the sound is markedly different, being loud and stridulous in character, especially inspiration.

The dyspnoeas dependent upon obstruction in the trachea or larger bronchi are similar, and although respiration be noisy, it lacks the stridulous character of laryngeal obstruction. The causes of respiratory difficulty are here usually enlargement of the bronchial or mediastinal glands from tuberculous disease or malignant growth and aneurysm of the aorta. The rarer causes met with in children are enlargement of the thymus gland or abscess in the posterior mediastinum, which may be secondary to disease of the dorsal vertebrae. The diagnosis of tracheal or bronchial dyspnoea can usually be made after a careful study of the physical signs in the two sides of the chest, special attention being paid to a comparison of the amount of an entering each lung and to any alterations in the quality of the respiratory murmur on the two sides. Additional evidence may also be obtained from the nature of the cough, especially if this is considered along with the results of laryngoscopic examination.

*Expiratory Dyspnoea*—This is specially characteristic of emphysema and asthma, and its nature can readily be determined on extra auscultation. In some cases it is laryngeal in origin, as, for instance, when due to a pedunculated subglottic tumour. A few cases have been recorded where pronounced expiratory dyspnoea in young children has been found post-mortem to be dependent upon enlargement of the bronchial glands. Such cases are exceptional, and while their explanation is not very satisfactory the fact of their occurrence should be borne in mind. The diagnosis of this form of dyspnoea can usually be arrived at from extra auscultation, and can be confirmed by finding the physical signs of emphysema and asthma.

Reference ought also to be made to the very rare form of dyspnoea, both inspiratory and expiratory, met with in cases of gross lesions of the muscles, e.g. pseudo-hypertrophic paralysis, and from certain injuries or diseases of the spinal cord.

A diagnosis can usually be arrived at by a careful investigation of each part of the respiratory tract. This will in many cases include

anterior and posterior rhinoscopy, digital exploration of the naso-pharynx, the use of the laryngoscope, and a careful physical examination of the chest, especially with a view of detecting physical signs dependent on pressure on the trachea, one or other bronchus, or other structures at the root of one lung. A useful point in differential diagnosis between laryngeal and tracheal dyspnoea lies in the fact, that in the former the larynx makes much respiratory movement, whilst in the latter it is stationary, however great the difficulty of breathing may be.

With regard to the severity of the dyspnoea in any given case, that depends not so much on the degree of stenosis as on the rapidity of its development. Another question calling for investigation is the permanent or intermittent nature of the difficulty. In the dyspnoea be laryngeal in site and constant, a paralytic lesion is suggested, whereas if intermittent it is suggestive of spasm, assistance in diagnosis will also be obtained from a study of the voice. The absence of voice impairment, however, does not negative a paralytic lesion, as a bilateral paralysis of the abductors would not be associated with any voice disturbance.

The foregoing remarks apply particularly to the usual run of cases where a definite obstruction exists to the inlet of air into the lungs or outlet of air from the lungs. Numerous other cases are encountered where the dyspnoea is largely produced or aggravated by a general medical cause such as cardiac disease, renal disease, asthma, emphysema, or acute lung disease, and in these affections we may find the exaggerated type of dyspnoea known as *orthopnoea*. Here the attitude is one of intense air hunger. The patient assumes the position which allows freest play to all the extraordinary muscles of respiration, and to this end he must be more or less in the upright posture, the position varying with the severity of the disease.

We must also bear in mind that mental and emotional disturbance may induce even in healthy subjects disordered respiration, and a tendency to this will be very much increased if there be present anything of the nature of an organic cardiac lesion. In this way may possibly be explained the occurrence of attacks of cardiac dyspnoea so frequently encountered. When dealing with dyspnoea associated with general cardio-vascular disturbance, the allied symptoms of palpitation and thoracic pain may be of assistance in diagnosis.

Attention should also be drawn to those cases of embarrassed respiration due to mechanical disturbance from intra-abdominal causes, either of a direct mechanical, or of a reflex nature.

*Dyspnoea in Children*—The same causes are at work as in the adult, but here temporary dyspnoea of a spasmodic nature due to spasm of the glottis is much more frequent. There is

also the relatively greater frequency of pressure on the trachea or bronchi from a tumour, glandular enlargement or abscess in the posterior mediastinum, and also anteriorly from the thymus gland, and in such cases the difficulties of respiration permanently present are liable to occasional exacerbations from spasms of the glottis. The symptoms in such cases are noisy breathing, metallic or croupy cough, the attacks of dyspnœa being usually nocturnal, and associated with other evidences of pressure.

Another factor of importance in the dyspnœa of children is the local muscular weakness of the chest wall present in rickets. Here the extraordinary muscles of respiration are readily called into play, and in marked cases of the inspiratory type, the movement of the muscles of the ala nasi and the indrawing of the lower intercostal spaces with inspiration form along with other general evidence a characteristic picture.

In older children the great frequency of obstruction due to adenoids should not be lost sight of.

**Dyssialia.**—Defective or faulty secretion of saliva.

**Dyspermatisim.**—Difficult or imperfect discharge of semen, e.g. from excessive erection of the penis (*hypertonic dyspermatisim*), from phimosis (*meatal dyspermatisim*), from urethral obstruction, want of force, epilepsy, etc. See SCROTUM AND TESTICLE, DISEASES OF (*Sterility*).

**Dyssynodus or Dysynusia.**—Difficulty in sexual connection.

**Dysthanasia.** Slow, lingering, and painful death, as opposed to *ethanasia*.

**Dystocia.**—Difficult, delayed, dangerous, or complicated labour, as opposed to *eutocia*, *fetal dystocia* is difficult labour due to some anomaly in the infant, in *maternal dystocia* the cause is to be found in the mother.

**Dystopia.**—Malposition or displacement.

**Dystrophy.**—A morbid state characterised by defective nutrition, due sometimes to disturbance of the trophic nerve supply of the parts (*neurotic dystrophy*). See MUSCLES, DISEASES OF THE (*Idiopathic Muscular Atrophy or Myopathy*).

**Dysuria.**—Difficult, painful, or incomplete micturition. See CHILDREN, CLINICAL EXAMINATION OF (*Urinary System, Dysuria*), GONORRHOICAL INFECTION, PUERPERIUM, PHYSIOLOGY (*Regulation of Bladder*), URINATION, DISORDERS OF (*Dysuria*).

**Ear.**—The diseases of the ear will be considered in the following articles. The affec-

tions of the **auditory nerve and labyrinth** have already been considered in vol. 1. pp. 327-336 (*q v*).

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See also AUDITORY NERVE AND LABYRINTH, BRAIN, PHYSIOLOGY (*Circulation*), CHIEK, FISSURE OF (*Varieties*), COUGH (*"Ear-Cough"*), DEAFMUTISM, ECZEMA (*Ears*), GALVANIC CAUTERY (*Diseases of Ear*), GLOSSO-PHARYNGEAL NERVE, GOUT (*Ear*), HEADACHE (*Causes, Reflex Irritation, Ear*), HEARING, ARTIFICIAL AIDS TO, LARYNX, CHRONIC INFECTIVE DISEASES (*Laryngeal Phthisis, Pain in Ear*), LIFE INSURANCE (*Diseases of Nervous System, Middle Ear*), LUNG, TUBERCULOSIS OF (*Complications, Nervous, Ear*), LUNGS, VASCULAR DISORDERS OF (*Pulmonary Embolism, Middle Ear Disease*), MALINGERING (*Varieties, Aural*), MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Symptoms, Ear*), MENTAL DEFICIENCY (*Abnormalities of Physical Formation*), MUMPS OR EPIDEMIC PAROTITIS (*Complications, Affections of Ear*), MYIASIS (*External, Aural*), NEPHRITIS (*Renal Cynchos, Hemorrhage into Tympanic Cavity*), PHYSIOGNOMY AND EXPRESSION (*Ears*), PHYSIOLOGY, THE SENSES (*Hearing*), POST-MORTEM METHODS (*Examination of the Brain, Middle Ear*), PURPURA (*Werthof's Purpura, Bleeding from Auditory Meatus*), RHEUMATISM, CHRONIC (*Clinical Features*), SCARLET FEVER (*Complications, Organs of Special Sense, Ear*), SKIN DISEASES OF THE TROCHS (*Boule, External Ear*), SYPHILIS (*Secondary Syphilis of the Ear, Tertiary Syphilis of the Ear*), TERNI (*Diseases of the Pulp, Pain in Ear*), TERATOLOGY (*Otocephaly*).

#### Ear, Examination of.

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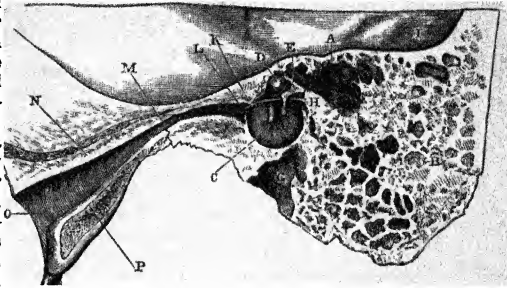
1. ANATOMY AND PHYSIOLOGY.—The anatomical subdivision of the organ of hearing into three parts—the external, middle, and internal ear—furnishes the clinician with a satisfactory basis upon which to investigate the nature of the affection in a case of deafness. Impairment in hearing may be due to causes which exist in one or more of these subdivisions or their adnexa, and the examination of the ear practically resolves itself into determining in which of them the lesion exists. In estimating the prognosis, too, and in considering the amenability of the affection to treatment, this anatomical basis serves a useful purpose. For these reasons, therefore, a brief sketch of the anatomy of the ear and its more intimate connections forms an essential introduction to its clinical examination.

The external ear consists of the pinna or auricle and the external auditory meatus, the former collecting the air vibrations, the latter conducting them inwards towards the tympanic membrane. The external auditory meatus is about one inch and a quarter in length, and has a general direction from without forwards, inwards, upwards, and downwards, so that it is not a straight canal. It is made up of two portions, an outer cartilaginous part about half an inch long, which is movable upon the deeper osseous portion; the latter measures about three-quarters of an inch in length. A well-marked angle or prominence exists in the antero-inferior wall of the osseous meatus and is the chief cause of the difficulty in the examination of the deeper parts. Beyond this point the lumen of the canal is again larger, and as its floor slopes downwards towards the tympanic membrane, a recess or sinus is formed in which foreign bodies may lodge. The mobility of the cartilaginous meatus is of the greatest assistance in the examination of the ear, as it permits of the canal being straightened to a considerable extent. For this purpose the auricle must be pulled upwards and backwards. The posterior wall of the osseous meatus has an intimate relation with certain of the mastoid cells, and sometimes when these cells are diseased perforation into the meatus occurs. The external auditory meatus is closed at its inner end by the tympanic membrane or drumhead, which separates it from the middle ear. This membrane is formed of three layers: the outer

cuticular layer is continuous with the skin lining the meatus; the middle is fibrous, an extension from the periosteum, and consists both of fibres radiating from the centre towards the periphery and also of circular fibres near the circumference; the inner mucous layer is a continuation on to the deep surface of the membrane of the mucosa lining the tympanic cavity. The blood-vessels pass to the membrana tympani along the handle of the malleus and thence radiate outwards between its layers.

Under the term *middle ear* there must be included the tympanic cavity, the Eustachian tube, the mastoid antrum, and mastoid cells, all of which are in direct communication with each other and, through the Eustachian tube, with the naso-pharynx.

The *tympanum*, *tympanic cavity*, *drum*, or *middle ear proper* is a small irregular space interposed between the external auditory meatus



Section through the whole extent of the middle ear (Barr). A, Antrum mastoidaleum; B, mastoid cells; C, inner surface of tympanic membranes at lower end of manubrium; D, head of malleus; E, body of incus; G, fossa for jugular bulb; H, pyramidal partition of bone separating the cavity of the tympanum from the antrum mastoidaleum; below the letter H a portion of the canal for the facial nerve is shown; I, dura mater; K, chorda tympani nerve; L, tympanic mouth of Eustachian tube; M, isthmus of Eustachian tube; N and P, cartilaginous walls of Eustachian tube; O, pharyngeal mouth of Eustachian tube.

and the internal ear or labyrinth. It is traversed from without inwards by a chain of ossicles, the malleus, the incus, and the stapes. The malleus or outermost bone is firmly attached to the tympanic membrane; the stapes, the most internal of the three, is fixed through its footplate to the membrane closing in the fenestra ovalis, which lies above the promontory at the upper and posterior part of the inner wall of the tympanum. The incus is the intermediate bone in the chain. The sound vibrations taken up by the membrana tympani are in this way conducted across the cavity to the labyrinth. It is necessary to draw attention to that part of the middle ear which lies above the level of the upper margin of the tympanic membrane, specially designated under the terms *attic* or *recessus epitympanicus*. This recess contains the head of the malleus and the greater part of the incus. The roof of the space is the tegmen tympani, a thin layer of bone, while its outer wall is formed

partly by the ledge of bone which constitutes the inner end of the roof of the external meatus, and below that by the upper part of the tympanic membrane known as Shrapnell's membrane. Certain ligaments attach these two ossicles to the walls of this recess, thus subdividing it, and in the event of suppuration occurring in it, rendering drainage difficult, and the affection therefore a more serious one. Such suppurations are usually associated with a perforation in Shrapnell's membrane. The relations of the facial nerve to the tympanic cavity are of great importance, and will be studied in detail when the operations upon the mastoid antrum are under consideration. It should be borne in mind here that, owing to an occasional congenital deficiency in the bony wall of the aqueductus Fallopii, facial paralysis may arise in the course of an acute otitis media. In chronic middle ear suppuration paralysis may follow caries of the bony wall of the Fallopiian canal.

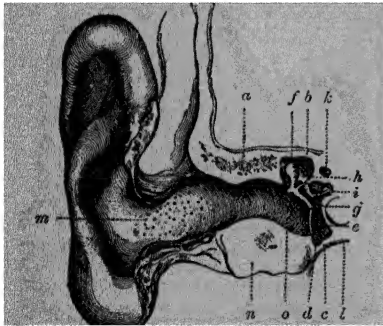
The *Eustachian tube* forms a direct communication between the naso-pharynx and the tympanic cavity, opening into the latter anteriorly and superiorly. Its pharyngeal orifice is situated on the external or lateral wall of the naso-pharynx, on a level with, and immediately

tympanic membrane, so that the membrane may fully respond to the sound vibrations. This air-pressure is maintained on the inner surface of the membrana tympani through the Eustachian tube, the pharyngeal orifice of which opens during swallowing by the action of the levator palati and salpingo-pharyngeus muscles. Any condition which tends to narrow or occlude the tube interferes with its proper function. This intimate anatomical association with the nose and throat renders the tube liable to share in the catarrhal affections of these regions, and it also serves as a canal along which infective processes may readily spread to the tympanum itself.

The *mastoid antrum and cells* lie behind the tympanic cavity, and are in direct communication with it posteriorly and superiorly through a short and somewhat irregularly-shaped passage, the mucous membrane being directly continuous from one cavity to the other. Posteriorly and inferiorly the antrum is separated from the tympanum by a ridge of bone, so that its floor lies below the level of the opening of communication; for this reason, drainage of pus from the antrum may be interfered with. Both the tympanum and antrum are roofed in by a thin plate of bone which separates them from the middle cranial fossa. The *mastoid cells* vary in their development, and for the most part lie superficial to the antrum. The connections of the middle ear are well shown in the above figures.

*The Internal Ear or Labyrinth.*—The third division of the auditory apparatus lies in the petrous temporal, internal to the tympanum, its outer bony wall being formed by the inner wall of the latter cavity. It consists of the osseous and membranous labyrinths with the terminations of the auditory nerve in the cochlea and vestibule; the cochlear fibres subserve the function of hearing, the vestibular are concerned with the maintenance of equilibrium. A more detailed account of the labyrinth and auditory nerve will be found under "Auditory Nerve and Labyrinth," vol. i. p. 327 *et seq.*

Clinical examination of the ear will be greatly facilitated by grouping the different anatomical parts above described under two heads: (1) The sound-conducting apparatus; (2) the sound-perceiving apparatus. By the *sound-conducting apparatus* we mean the auricle, the external auditory meatus, the membrana tympani, the tympanum and chain of ossicles, including the insertion of the footplate of the stapes in the oval window. The *sound-perceiving apparatus* consists of the terminations of the auditory nerve in the cochlea, the nerve-trunk itself, the various tracts in the brain, and the auditory centre. When the lesion involves the conducting portion, it is frequently spoken of as producing "obstructive deafness," and when the perceiving portion is at fault, the term "nerve deafness" is employed. It is essential that the



Vertical section of the external meatus, membrana tympani, and tympanic cavity (Politzer). *a*, Cellular apices in the superior wall of the meatus, connected with the middle ear; *b*, roof of tympanic cavity; *c*, inferior wall; *d*, tympanic cavity; *e*, membrana tympani; *f*, head of the malleus; *g*, handle of the malleus; *h*, incus; *i*, alapes; *k*, canal Fallopii; *l*, fovea jugularis; *m*, glandular orifices in the external meatus (right ear).

behind, the posterior end of the inferior turbinate body. The direction of this orifice is downwards as well as inwards, while posteriorly and superiorly its circumference presents a well-marked prominence or cushion. Special reference must be made to the Eustachian tube, owing to the important part which it plays both in the physiology of hearing and in the pathology of deafness. It is essential for the proper function of hearing that equal air-pressure should be maintained on both surfaces of the

examination should be conducted along such lines as will lead to the localisation of the affection in one or other, or in both, of these general subdivisions

2 SYMPTOMATOLOGY.—The chief symptoms and signs in connection with disease of the ear may be briefly stated to be (a) disturbance in hearing, (b) tinnitus, or subjective noises in the ear, (c) pain, (d) vertigo or giddiness, and (e) discharge from the ear

(a) *Disturbances in Hearing*.—The onset of deafness may be rapid, or it may be more or less gradual. A sudden onset may result from a collection of wax in the external auditory meatus, or it may be due to a labyrinthine effusion. In all cases of sudden deafness, inquiry should be made as to the previous existence of defective hearing. If the onset is very gradual, it is often difficult for the patient to give any definite statement as to the exact time and mode of its origin. In all cases coming under observation, an examination of both ears should be made, though the patient may complain of only one of them. Careful testing may reveal incipient mischief where none was suspected, and prognosis may thereby be influenced. The more frequent occurrence of the exanthemata in children, and the greater tendency for acute inflammatory processes to pass from the naso-pharynx to the middle ear, render that organ more liable to inflammatory attacks in the young. In adult life, on the other hand, the chronic catarrhal affections of the tympanum are more common, while in advancing years the auditory nerve tends to react less readily to sound vibrations. Heedily undoubtedly plays a part in the causation of some forms of deafness, thus necessitating a careful inquiry into the family history in such cases. The presence or absence of certain anomalies in hearing should be inquired into, such as hearing better in a noise (*paracusis Willisii*), or double hearing (*diplacusis*). The patient may complain that certain sounds produce almost a painful impression, *hyperæsthesia acustica*, but this is a less common anomaly.

(b) *Tinnitus Aurium or Subjective Noises in the Ear*.—Subjective sensations of sound are very frequently complained of, these sounds have no objective cause outside the body, and they vary alike in their character and their intensity. It should be borne in mind that they may be intra-aural in origin and due to changes of tension in the ear itself, again, they may be reflex, the reflex cause being found in a diseased tooth, or possibly excited by gastric disturbance, lastly, and not infrequently, they are of vascular origin, and may be due to changes in the condition of the walls of the blood-vessels, as in Bright's disease, to changes in the circulation in heart disease, and to changes in the quality of the blood itself, as in anemia. It is sufficient merely to indicate these points here, so that the attention of the aurist may not be confined

entirely to the ear itself in the elucidation and treatment of this most disagreeable complaint.

(c) *Pain*.—Pain is a marked symptom in acute inflammations of the external and middle ear, when complained of in association with chronic suppurative of the latter cavity it becomes a symptom of considerable gravity. It must not be forgotten that pain may be referred to the ear in association with carious teeth, objective examination of the ear and of the teeth will, as a rule, clear up this point. Pain aggravated on moving the jaw, on pressure upon the tragus, or on pulling the auricle suggests the presence of a meatal inflammation, on the other hand, if deep-seated, intermittent in character, being worse at night, and aggravated by blowing the nose and coughing, it is more symptomatic of an acute otitis media. When pain is associated with discharge from the ear, attention should be paid to the condition of the mastoid process, and its presence may be elicited by careful pressure upon the bone.

(d) *Vertigo or Giddiness*.—This symptom may be sudden and severe, as in the typical Ménière's disease of the labyrinth, or it may be very slight in character. Anything which will alter the tension of the labyrinthine fluid may cause some degree of vertigo, hence the pressure of wax upon the tympanic membrane, an indrawn condition of the membrane, or the presence of fluid in the tympanic cavity may exert sufficient pressure through the chain of ossicles upon the fenestra ovalis as to increase the tension of that fluid. Giddiness preceded by nausea may be due to gastric disturbances, giddiness followed by nausea suggests, on the contrary, its otitic origin.

(e) *Discharge from the Ear*.—Discharge from the ear varies in its character, it may be thin and serous, often containing flakes of epithelium, as in the eczematous conditions of the meatus, it may be mucopurulent and stringy, or purulent and thick, sometimes it is tinged with blood, an appearance which should always suggest the presence of granulations or polyp. Discharge from the ear may be meatal in origin, as in eczema or in acute inflammatory conditions, but as a rule its source lies in the middle ear, and it drains through a perforation in the tympanic membrane. It may be extremely offensive in character, and in every case of aural discharge the examiner should make it a routine practice to smell the speculum on its withdrawal. The factor which so frequently accompanies a chronic middle ear suppuration is pathognomonic of that condition. The patient's statement as to the cessation of an aural discharge should never be accepted until a careful examination of the ear has been made. If the examiner should fail to satisfy himself of its presence by mere inspection through the speculum, he should not fail to introduce into the bottom of the external meatus a wick of absorbent wool, and on its withdrawal

examine it closely for any indication of moisture or fester

**3 INVESTIGATION OF THE FUNCTION OF HEARING.**—We have already indicated in the anatomical sketch of the ear that impairment of hearing may be due to causes existing in the sound-conducting or sound-perceiving apparatus, or in both. While an objective examination of the ear will assist in determining the site (and nature) of the lesion in the former, the possible existence of a labyrinthine affection can only be accurately judged by carefully testing the hearing function. Hence such tests serve the double purpose of estimating not only the degree of deafness that may be present, but also of localizing the seat of its origin.

Sound sensations may reach the auditory nerve terminations through two channels, the external auditory meatus and chain of ossicles, *i.e.* by air-conduction, and through the bones of the skull, *i.e.* by bone-conduction. Certain simple methods are employed for testing audition through both these channels, these are the watch, the voice, and the tuning fork. While carrying out this part of the examination care must be taken to eliminate every source of error, if reliable observations are to be made. It is very necessary that the different tests be applied before any treatment is carried out, and that the results obtained by these tests be at once carefully noted in writing, the date of the first as well as of later examinations should be registered, and the surrounding conditions on subsequent occasions should be as nearly as possible similar to those which existed in the first instance. The same test apparatus should be again employed. In every case the hearing power of both ears should be ascertained, although the patient may only complain of deafness in one of them.

Before proceeding to apply the various tests it is advisable to introduce the speculum and ascertain whether any obstruction exists in the external auditory meatus of the nature of wax, etc., so that a needless repetition of the tests may be avoided by a preliminary removal of the obstruction.

(a) *The Watch Test*—The distance at which the tick of any given watch is heard by the normal ear is previously ascertained by experiment upon a number of healthy ears, so that a definite standard for comparison is thus provided. This distance may be recorded in inches.

*Method*—The patient is placed with one ear directed towards the examiner, while the other is closed by the introduction of a finger. The eyes may with advantage be closed, a precaution which is especially necessary in the case of children. The examiner, holding a yard measure in one hand, gradually brings the watch towards the ear under examination from a point outside the range at which the tick is

perceived by a normal ear. Care should be taken that the watch does not touch the foot-rule if the latter is in contact with the bones of the head. The patient is directed to state at once when the tick becomes audible, after two or three control experiments have been made, the distance is noted and the measurement registered in inches. This may be expressed in terms of a fraction, whose denominator represents the normal hearing distance of the watch, *e.g.* 30 inches, while the numerator expresses the distance in any given case, *e.g.* 5, *i.e.*  $\frac{5}{30}$ . If the watch be heard only on contact or on pressure upon the skull, it may be expressed as  $\frac{1}{30}$ , while if the tick is not perceived at all, it may be registered as  $\frac{0}{30}$ . The same procedure is then carried out with the other ear.

(b) *The Voice Test*—In estimating the hearing distance by means of the human voice, the whisper and the ordinary conversational voice are employed, these may be variously modified, so that low and loud whisper, low, ordinary conversational, and raised voice may be used as the examiner sees fit. The result is recorded in feet.

*Method*—The patient remains seated sideways as before, it being very necessary that the movements of the examiner's lips should not be observed, the ear of the opposite side is again closed with the finger. The observer retires across the room, having directed the patient to repeat at once the words which he succeeds in hearing. If he should fail to hear anything at this distance, the examiner gradually approaches until he comes within hearing distance. This is carried out both with the whispering and with the ordinary conversational voice. The other ear is then examined and the results are again registered in feet. With regard to the words used for this purpose, it is perhaps advisable to employ single words, the patient in each instance repeating them when heard. When sentences are spoken or questions asked, the perception of one or two words only may give the key to the whole, and in this way a guess may prove correct and a false estimate be thus obtained. The numerals supply suitable syllables if too much repetition be avoided. The necessity of maintaining a uniform pitch in the use both of the whispering and of the speaking voice is evident, and facility in acquiring this will be obtained by practice.

*Hearing Tests in Children*—In children it is often difficult, and in the very young even impossible, to obtain satisfactory answers either to the watch or voice tests. In the majority of cases the former is quite unreliable. It may sometimes suit all practical purposes to ask a few simple questions in ordinary tones and note whether they are responded to. On the other hand, it may be necessary to instruct the parents as to what is wanted, so that they may

examine the child at home in the absence of any stranger. In the case of very deaf children, for the purpose of ascertaining whether any sound perception exists at all, such tests as clapping of the hands, the ringing of a bell, and blowing of a whistle may be employed. The source of such sounds must not be observed by the child, whose attention should be engaged, and whose expression should be watched by the parent or attendant. At the same time no current of air produced by these acts should fall upon the patient's head or face, nor must any mechanical vibration reach him, as a source of fallacy is thus introduced.

(c) *The Tuning-Fork*—It is unnecessary to enter fully into a consideration of the tuning-fork tests, as a detailed description has already been given under "Auditory Nerve and Labyrinth," vol. 1, p. 327. It will suffice here to briefly recapitulate the different points. The vibrations of a tuning-fork are conducted to the nerve-endings in the labyrinth, through the external auditory meatus and chain of ossicles, and also through the cranial bones. The former is spoken of as "air-conduction," the latter as "bone-conduction" of tuning-fork vibrations. In a normal ear these vibrations are somewhat better heard by air-conduction. If in a deaf person the vibrations of a tuning-fork are perceived more loudly when the fork is applied to the skull than when it is held in front of the ear, "obstructive deafness," or a lesion in the sound-conducting apparatus is indicated; in other words, there is "increased bone-conduction." On the other hand, if the vibrations are better heard in the deaf ear when the fork is held in front of the meatus, "nerve deafness" is indicated, i.e. there is "diminished bone-conduction." These facts may be more briefly expressed as bone-conduction + ( $BC > AC$ ) or bone-conduction - ( $AC > BC$ ) (Weber's test). In a normal ear when a vibrating tuning-fork is placed on the mastoid process and kept in contact until the sounds are no longer heard, they again become audible when the fork is held opposite the meatus (Rinne's test positive). If in a deaf ear the tuning-fork, after ceasing to be heard on the mastoid, be held opposite the meatus and is not again heard, Rinne is negative, and a lesion in the sound-conducting apparatus is indicated. On the other hand, if in a deaf ear the tuning-fork vibrations are again heard in front of the meatus, Rinne is positive, and a lesion of the sound-perceiving apparatus is indicated. These facts may be more briefly expressed as Rinne - and Rinne +. (Gardner Brown's and Schwabach's tests may be useful as supplementary tests.)

*Other Tests occasionally employed*—(Gardner Brown's)—In the normal, if the base of a medium vibrating tuning-fork is held on the mastoid process, it ceases to be heard at the same time that a trained finger and thumb

cease to feel the vibrations. In middle ear disease the patient will hear the sound some time after the observer has ceased to feel the vibrations. In internal ear affections the patient ceases to hear the fork some seconds before the vibrations have ceased to be felt.

Schwabach's—In using this test the observer compares his own bone-conduction, which must be normal, with that of the patient. If the vibrating tuning-fork placed on the mastoid process of the patient has ceased to be heard by him, but is still heard when placed on the mastoid process of the observer, labyrinthine disease is indicated; on the other hand, if the patient hears the fork after the observer has ceased to do so, the existence of middle or external ear disease is suggested.

*Tests for Range of Tone Hearing in Labyrinthine Disease*—In addition to the facts already elicited by the use of the tuning-fork, further information can be obtained by testing the appreciation of the ear for sounds of different pitch. It is generally admitted that when the impairment of hearing is most marked for high notes—the low tones being relatively well heard—the labyrinth and sound-perceiving apparatus is at fault. On the other hand, when the impairment of hearing is most marked for low notes—the high tones being relatively well heard—the lesion is probably to be found in the sound-conducting apparatus. For this purpose a series of tuning forks of varying pitch may be used, or one fork provided with metal clamps, which may be moved up and down the limbs, the deeper tones being produced when the clamps are fixed towards the free ends of the fork and *vice versa*. By the use of Galton's whistle, similar variations in pitch are obtained; care should be taken that the patient does not confuse the blowing sound produced by the whistle with the true whistling note. In the absence of such instruments as these, an attempt may be made to form some estimate of the relative power of appreciating high and low tones by the use of letters of different pitch. According to Wolfe, R represents a sound of very low pitch, S, on the other hand, is of high pitch. In internal ear deafness the voice may be heard relatively more distinctly than the higher note of the watch tick. Various musical instruments may also be used for testing gaps in the range of hearing.

*Simulated Deafness*—Various devices are employed for the detection of feigned deafness. If the hearing in the affected ear is said to be *impaired*, the patient should be carefully tested when blindfold. If *total* deafness, on the other hand, be feigned in one ear, the ear pieces of a binaural stethoscope should be introduced into the patient's ears, the end inserted into the ear in which hearing is said to be present having been previously plugged with a piece of wood. If the patient repeats words which are whispered

into the cup-shaped end of the stethoscope, the true nature of the condition becomes evident. Valuable information may be obtained by the use of the tuning-fork if the patient is ignorant of the true results of the different tests. When complete bilateral deafness is feigned, the diagnosis becomes more difficult, and it may be necessary to adopt the ruse of attempting to awaken the malingerer from sleep.

**4. OBJECTIVE EXAMINATION OF THE EAR.**—Before proceeding to examine the deeper parts of the external auditory meatus and the tympanic membrane, the surgeon should make a simple inspection of the auricle and adjacent parts, because much useful information may be obtained by a preliminary examination of this kind. The patient's features may present the character so frequently spoken of as the "adenoid facies," the result of nasal obstruction, or the existence of facial paralysis may be detected on one or both sides. Enlargement of the glands in front of, below, or behind the auricle should be noted and their condition more exactly determined by careful palpation. In children an otorrhoea associated with enlarged lymphatic glands and facial paralysis strongly suggests the tubercular nature of the affection. If there is any undue prominence of the auricle the cause should be investigated. Redness of the skin, oedema and swelling over the mastoid process, and the presence of a sinus or cicatrix in the same region should not be overlooked, and the significance of such signs must be duly estimated. Malformations of the auricle and tumours may present themselves, while eczema confined to the pinna or in association with a like condition of the auditory meatus may be readily detected on inspection. It must not be forgotten that with suitable illumination the outer portion of the meatus can be examined without the introduction of the speculum. Such an examination, aided by pulling the auricle upwards and backwards and by gently drawing the tragus forwards, should be carried out as a routine practice. In this way the patient may be saved needless pain or discomfort, such as might be caused by the insertion of the speculum into an inflamed meatus, at the same time boils, eczema, pus, and even the presence of a polypus or plug of wax can be in this way readily detected.

*Examination with the Aid of the Aural Speculum.*—*Source of Light.*—Good illumination is essential for a proper examination of the ear. The deeper parts of the external auditory meatus and the tympanic membrane are most satisfactorily examined by *reflected light*, i.e. by the indirect method. The source of the light will vary according to circumstances, if bright daylight can be obtained, no better source of illumination need be wished for. The actual rays of the sun may prove somewhat trying to the observer's eye, and care must be taken to

prevent the rays falling directly into the ear from a concave reflector, otherwise the parts may be burnt. Of the artificial sources of light, mention may be made of the Welsbach incandescent gas from an argand burner, the electric and oxyhydrogen light. A simple oil lamp or candle may prove quite satisfactory in default of any other means.

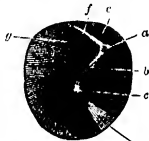
*The Reflector.*—The small aural reflector in common use has a diameter of about three inches and a focal distance of five inches, and is perforated with a small central aperture. It should not only be provided with a handle, but should be capable of being easily adapted to a forehead band or spectacle frame, so as thus to leave both hands of the examiner free if necessary.

*The Aural Speculum.*—Numerous forms of specula have been introduced, and it would be useless to enumerate the varieties here. The operator soon accustoms himself to the use of one form, which he learns to regard as superior to any other. Only the simple tubular speculum should be employed. A dilating speculum for the ear has no advantages, and only causes the patient pain and discomfort. A set of specula consists of four, the silver instruments have this advantage, that they can be boiled, vulcanite specula are useful when caustics are applied to the ear.

(a) *Technique of the Examination.*—A satisfactory examination of the deeper parts of the ear depends largely upon good illumination and correct manipulation of the pinna and speculum. The patient is seated with the ear to be examined turned away from the source of light, his head being slightly inclined towards the opposite shoulder. The surgeon, sitting or standing, holds the reflector lightly in one hand, in the right hand for the right ear, in the left hand for the left, placing the upper margin against his forehead, and tilting it to the angle required for throwing the best possible light upon the parts. His eye, the right or left as the case may be, must be placed directly behind the central aperture of the mirror. A medium-sized speculum, held by its margin between the index finger and thumb of the other hand, is now gently inserted into the cartilaginous meatus, and at the same time the upper part of the pinna is grasped between the middle and ring fingers of the same hand and pulled upwards and backwards, thus rendering the canal as straight as possible. The speculum can now be insinuated a little farther, slight rotation assisting the movement, due care, however, being taken that it is not inserted as far as the bony meatus. As a complete view of the whole tympanic membrane is not always obtained in one field, the outer end of the speculum should be moved backwards, forwards, upwards, and downwards, so that the various areas may be brought into view. The patient is then

turned round and the other ear is examined in a similar manner. In every case both ears should be inspected, although only one may be complained of. Certain preliminary difficulties present themselves, but a little practice soon enables the observer to obtain the maximum amount of light, to inspect the parts through the central aperture of the mirror, and to straighten the auditory meatus by pulling the pinna sufficiently upwards and backwards. Should wax or other cause of obstruction interfere with the examination of the deeper parts, the ear should be syringed. If the canal is too narrow to admit of the insertion of a medium-sized speculum, a smaller one may be substituted, but the routine use of small specula should be avoided, as the field is thus curtailed and the amount of light considerably reduced. If it is thought desirable to remove a piece of wax or epithelial flakes with the ear forceps, or should it be necessary to use the probe, the reflector must be attached to the forehead band or spectacle frame, so that the left hand becomes free to manipulate the speculum and pinna, while the forceps or probe is held in the right. Occasionally a reflex cough is induced by the insertion of the speculum into the meatus.

(b) *The Tympanic Membrane.*—The tympanic membrane (*membrana tympani*) or drumhead is of a bluish-grey colour with a somewhat polished surface, presenting a distinct contrast both in colour and apparently in consistence to the white skin lining the deeper part of the external meatus. Concave as a whole, on its outer or mental surface it occupies an oblique plane, so that the superior and posterior parts lie nearer the observer's eye than the anterior and inferior. The handle of the malleus (*manubrium mallei*) is visible on the outer surface of the membrane as a slender ridge of a whitish-yellow colour



Outer aspect of right tympanic membrane, double the natural size. *a*, Short process; *b*, middle of manubrium; *c*, umbo; *d*, cone of light; *e*, *membrana flaccida*; *f*, posterior fold; *g*, long process of incus shining through the membrane (Barr).

traversing the membrane from above downwards and backwards to a point a little below the centre. At the upper end of the handle there is a distinct white knob-like projection, the *short process* of the malleus. The lower end, on the other hand, forms a slight expansion often distinctly visible, which is situated at the umbo or point of greatest concavity of the membrane. Passing downwards and forwards from the umbo towards the circumference of the drumhead may be seen the triangular bright spot or *cone of light*, with its base towards the periphery. This triangle of light often forms a conspicuous landmark in the field of vision, but it is subject to considerable alterations both in shape and

even on healthy membranes; as it is produced by the reflection of the rays of light from that part, and as its shape is dependent upon the plane of obliquity of the membrane, very slight changes may influence its shape and size. If the examination be now directed above the short process of the malleus, Shrapnell's membrane (*membrana flaccida*) comes into view, differing structurally from the rest of the membrane in that the middle fibrous layer is absent. An anatomical perforation may exist in this area. Passing forwards and backwards from the short process of the malleus to the periphery are seen two elevations or folds of membrane, the *anterior* and *posterior folds*. They are caused by the projection of the short process, and with it constitute the lower limit of Shrapnell's membrane. Sometimes in normal conditions the long process of the incus may be seen through the membrane behind the handle of the malleus, and running more or less parallel to it. If the membrane be very transparent or atrophied, other intra-tympanic structures may become visible, such as the incus-stapes joint, and even the chorda tympani nerve. For descriptive purposes the membrane may be subdivided as follows:—A primary division into two by an imaginary line prolonging the handle of the malleus downwards to the periphery and forming a larger posterior and a smaller anterior segment; the subdivision of each of these segments again by a line bisecting the former one, so that four divisions result, an anterior superior, anterior inferior, posterior superior, and posterior inferior quadrant. The various lesions may be located and described as existing in one or more of these areas.

*The mobility of the tympanic membrane* should be tested. This may be done in one or more of the following ways:—(a) By inflation of air through the Eustachian tube by the method of Valsalva, (b) by inflation by Politzer's method or by means of the Eustachian catheter, and (c) by the use of Siegle's speculum. Inflation of the tympanum will be described presently; it is only necessary to say here that the tympanic membrane must be carefully observed through the speculum during the process of inflation, and the presence or absence of movement noted. Should Politzer's method or the Eustachian catheter be used, an assistant is required to carry out the inflation.

Siegle's pneumatic speculum somewhat resembles a large ear speculum, but its expanded end is covered with a piece of glass placed obliquely, while to an aperture in one side of it there is attached a tube and indiarubber ball. The narrow extremity of this speculum must be made to accurately fit the external auditory meatus so that the latter along with the speculum forms an air-tight cavity. The examiner with the reflector on his forehead observes the tympanic membrane, while he

alternately compresses and relaxes the india-rubber ball, this compression and exhaustion of the air column in the meatus causes the different mobile parts of the membrane to move. By careful observation the actual movements of the tympanic membrane may be seen, or a change in its position may be indicated by the appearance in one or more situations of bright reflecting spots. If the membrane moves outwards on inflation, but almost at once sinks back again, although the patient has not swallowed, it is probable that adhesions exist between it and the inner wall of the tympanum.

*Pathological Conditions of the Tympanic Membrane—Variations in Colour*—The tympanic membrane may vary to some extent in its colour, polish, and transparency consistently with a normal standard of hearing, the appearances being slightly modified by the intra-tympanic structures which may be seen through it. Behind the umbo a whitish area may be due to the promontory on the inner wall of the tympanum, while behind the handle of the malleus the long process of the incus or the incus-stapes joint may be visible through the membrane as greyish-white structures, in the posterior inferior quadrant a dark area may signify the niche leading to the fenestra rotunda. In the more advanced years of life there is a tendency for the membrane to become whiter and less polished. Such normal conditions, however, may be exaggerated and have a pathological significance. Thus in general atrophy of the membrane, the incus, stapes, and promontory may become very apparent. If circumscribed atrophic patches exist, they appear as dark transparent areas which are not distinctly demarcated from the surrounding membrane, on inflation, or when seen through Siegle's speculum, the atrophic areas show great mobility, and may bulge freely into the meatus. A cicatrix, the evidence of a healed perforation, usually presents itself as a dark transparent area, often difficult to distinguish from a patch of atrophy, but it is more sharply defined from the surrounding healthy membrane than is the atrophied portion. Owing to the transparency of the cicatrix, it may only be possible to differentiate it from a perforation by observing the result of rarefaction of the air in the meatus with Siegle's speculum, if a cicatrix be present, it will be seen to move outwards when the india-rubber ball is relaxed, in the case of the perforation no movement is observed. *Thickening of the membrane* varying in degree and extent may show itself in the form of opaque patches, or the whole membrane may be more or less white and without its natural polish. *Calcareous deposits* may form in its layers and be evident as white chalky areas, sometimes of a crescentic shape. If touched with the probe their calcareous nature is at once demonstrated. The *inflamed membrane* presents different appear-

ances, according to the degree and stage of the inflammatory process. The congestion may be limited to the vessels about the handle of the malleus and its short process, or they may be seen radiating outwards across the membrane as a number of irregular pink lines. On the other hand, the whole membrane and the deeper part of the osseous meatus may present the appearance of deeply inflamed skin. The presence of *fluid in the tympanum* may be indicated by a yellow appearance of the lower part of the membrane, the discoloration being bounded above by a dark line stretching across the drumhead. The membrane may be bulged outwards into the meatus either in whole or part by exudation behind it, so that it occupies a plane nearer to the observer's eye than that which the normal membrane has. It is important to learn to appreciate these alterations in the plane of the tympanic membrane. *The indurated membrane* so frequently met with is characterised by the prominence of the short process of the malleus and of the anterior and posterior folds, the handle of the malleus is drawn backwards and inwards to a varying degree so that it becomes foreshortened, and in some cases assumes an almost horizontal position, the cone of light may entirely disappear or be visible merely as one or more small reflecting spots of light. The membrane as a whole lies on a deeper plane than normal, and presents a more distinctly concave appearance. A *perforation* may be present in the tympanic membrane, as a rule it is single, but sometimes more than one exists, they vary in size from a small pin head to a complete destruction of the whole membrane. A portion of, or nearly the whole of, the inner wall of the tympanum may be visible through the perforation, and experience will lead the observer to appreciate the distinction between the plane of the membrane and the deeper plane of the inner wall, in these cases further assistance in diagnosis may be derived from the use of the probe, the hard consistence of the bony wall forming a distinct contrast to the more yielding nature of the membrane itself. The diagnosis of a perforation associated with fluid in the tympanum may be further assisted by inspecting the ear during inflation, when bubbles of air mixed with secretion may be observed. When the perforation is very minute it may be impossible to see it, but one sign which is almost pathognomonic of the condition is the presence of one or perhaps more pulsating spots of light, distinctly visible on inspection. The secretion in contact with the inflamed mucous lining of the tympanic cavity rises and falls with every heart-beat, it is very doubtful if this phenomenon be visible to the eye when the membrane is intact. *Granulations* may be recognised as red fleshy masses varying in size, they are of soft consistence and readily bleed when touched, when large they constitute



aural polypi, recognised by their closer proximity to the speculum, their mobility, and the fact that the probe may be passed round them. In all cases of suspected perforation the precaution should be taken of smelling the speculum, the presence of a foetid odour, so significant of *chronic* middle ear suppuration, and therefore of an existing perforation, is a valuable diagnostic aid. In these cases another speculum should be employed for the examination of the opposite ear, otherwise there is room for a possible fallacy in the second instance.

5. **EXAMINATION OF THE NOSE, NASO-PHARYNX, PHARYNX, AND FAUCES.**—Owing to the intimate anatomical relation which exists between the middle ear and the naso-pharynx through the medium of the Eustachian tube, it is essential that a careful examination of the throat and nose should be made in all cases, indeed, this inspection should be a routine practice. It is necessary both on diagnostic and therapeutic grounds, and the aurist should make himself thoroughly acquainted with both the normal and pathological appearances of these parts, and become thoroughly efficient in the necessary technique. Such inspection is best made at this stage, before inflation of the tympanum is practised. Examination of the nose by anterior and posterior rhinoscopy will be described later (see "Nose"). In some cases it may be necessary to supplement the procedure by a digital exploration of the naso-pharynx, and in children this may prove the only way of arriving at a satisfactory conclusion with regard to the condition of the post-nasal space.

Inspection of the fauces may reveal the presence of enlarged tonsils, when these are found in children, especially when associated with large pale granules on the posterior pharyngeal wall, the probability of the coexistence of adenoid vegetations is suggested. A difficulty or sluggishness in the elevation of the soft palate on phonation favours the same conclusion being drawn. It is only in very rare instances that the post-nasal growths themselves can be observed by simple inspection of the pharynx. The presence of an excess of secretion upon the posterior pharyngeal wall may signify the existence of naso-pharyngeal catarrh. In the examination of the anterior nares special attention should be paid to the presence of spurs, ridges, or septal deviations or other obstructive agents which might interfere with the passage of the Eustachian catheter, should that be found necessary at a later stage.

6. **EXAMINATION BY INFLATION THROUGH THE EUSTACHIAN TUBE.**—The introduction of a current of air into the middle ear through the Eustachian tube is a most valuable and essential aid both in the diagnosis and treatment of many ear affections. It is on account of its therapeutic value that due care should be taken to accurately note the patient's power of hearing

before any method of inflation be tried. We have already incidentally referred to it in testing the mobility of the tympanic membrane, but it finds a more extended application than that. In diagnosis, the permeability of the Eustachian tube, the presence of secretion at the Eustachian orifice or in the tympanum, and the existence of a perforation in the membrana tympani may all be determined by this means. In estimating prognosis, too, it is a reliable guide, and as a means of treatment it is of the greatest value. It is necessary, therefore, to be thoroughly conversant with the technique of the various procedures employed for this purpose.

Three methods are in common use—(1) *Valsalva's method*, (2) *Poltzer's method*, (3) *inflation through the Eustachian catheter*.

(1) *Valsalva's Method*.—By this means auto-inflation of the middle ear can be practised. The patient is directed to hold the nose tightly between the finger and thumb, to close the mouth and forcibly expire, at the same time puffing out the cheeks. If the Eustachian tubes are pervious, they provide the only outlet for the air which is thus forced through them and impinges upon the inner surface of the tympanic membrane. As already indicated, during this procedure the observer inspects the drumhead through the aural speculum. It must be borne in mind that syncope may be thus induced in persons with a weak heart, owing to a too forcible expiration with all the outlets closed. This method of inflation has a somewhat limited application.

(2) *Poltzer's Method*.—In order to inflate the tympanum by this method, a Poltzer's bag and an auscultating tube are necessary. The bag may have an air capacity of six or eight ounces, or even more, and should be provided with a valve, its nasal end should be fitted with a nose nozzle, or what is better, the extremity should be covered before its insertion with a small piece of rubber tubing. This has the double advantage of being soft, and further, of securing greater cleanliness, as a fresh piece may be used for each patient. For auto-inflation it is perhaps more convenient to have a vulcanite nozzle attached to the bag by a piece of rubber tubing two or three inches in length. The examiner should make it a routine practice to auscultate during inflation, as in this way he may satisfy himself as to whether air enters the tympanic cavity, and as to the character of the sound produced. The patient's statement as to the first point is not reliable. Auscultation is carried out by the aid of a long rubber tube, each end of which is provided with a small ear-piece, one coloured black for insertion in the external auditory meatus of the patient, the other white for the surgeon's ear. It is neither necessary nor advisable that the patient should hold this tube in his ear, adventitious sounds

may be produced by the friction of the fingers upon it.

The operator stands in front of his patient, having connected his ear to that of the patient by means of this tube, he directs him to take a sip of water and to keep it in his mouth until ordered to swallow it. He then grasps the air-bag in his right hand, inserting its nozzle just within one nostril, with the finger and thumb of the left hand both nostrils are firmly compressed. The position of the bag when held in the right hand is oblique, lying more or less in the long axis of the external nose. The patient is now directed to swallow the water—and it may be necessary to say this in a loud tone,—and simultaneously with the movements of the larynx during this act the bag is sharply and forcibly squeezed. The operation may be repeated either through the same nostril or through the opposite one, the surgeon's ear being now connected with the other ear of the patient. In Politzer's method both Eustachian tubes are inflated simultaneously.

Certain modifications of this method have been suggested, thus Holt, instead of asking his patient to swallow water, directed him merely to blow out his cheeks while the mouth was kept shut. While this procedure is considered less disagreeable by many patients, the entrance of air into the tubes is not assisted as it is during the act of swallowing, the orifices being then opened by muscular action. Holt's modification, however, has this advantage that no adventitious sound is produced such as accompanies swallowing, nor is the rush of air into the tympana quite so forcible. Gruber suggested phonation of the word "huck" (pronounced "hook"). Lucæ directed his patient to utone the vowel sound "ah" during the act of inflation. In both these modifications the soft palate is raised and the naso-pharynx is thus shut off. It may be noted here that in the case of young children, where it is useless to attempt to give any instructions, the act of crying is of the greatest assistance.

### (3) Inflation through the Eustachian Catheter

—For this operation the examiner requires in addition to Politzer's bag and the auscultating tube, a Eustachian catheter. Silver or vulcanite instruments may be used, the former possessing this great advantage that they may be sterilised by boiling, the greater pliability of the vulcanite instrument, however, renders it more easy to manipulate in the nose. The catheters vary in length, in the shape of their curve, and in the size of their lumen, so that various anatomical difficulties may be thus overcome. In selecting an instrument in any given case for the first time, it is perhaps advisable to make use of one which has a full curve and a medium-sized lumen. The small metal ring on the proximal end of the catheter indicates the direction of its point, before inserting the catheter the

surgeon should attach the Politzer bag to the funnel-shaped proximal end and blow air through it, thus satisfying himself both as to its patency and to the absence of any liquid in its lumen.

If during the previous examination of the nose the anatomical condition suggested a difficulty in the introduction of the catheter, the operation should be commenced with the aid of the speculum and artificial illumination, otherwise the patient is seated with his face turned towards the light.

**1st Stage**—The surgeon, facing his patient, first connects his own ear by means of the auscultating tube with that of the patient. Politzer's bag is placed under the left arm, with the large end directed forwards so that it can be readily grasped in the right hand when required. The catheter is then held lightly between the index finger and thumb of the right hand, care being taken not to conceal the metal ring in so doing. The inner edge of the left hand is laid upon the patient's forehead, while the tip of the nose is gently tilted up with the thumb of the same hand, thus removing the obstructing ledge at the junction of the cartilaginous and osseous floor. The beak of the catheter is now inserted point downwards, the right hand being at this stage depressed below the level of the patient's chin. As soon as the point of the instrument has slipped over the elevation of the floor just alluded to, the right hand is at once raised and the catheter brought into the horizontal position, when it is pushed backwards along the inferior meatus of the nose until the convexity of the curve is felt to come in contact with the posterior wall of the naso-pharynx. It is very necessary that the hand should be raised as directed so that the point of the instrument is kept in contact with the floor of the nose, otherwise it may pass upwards into the middle meatus and there prove a source of annoyance both to the patient and surgeon.

**2nd Stage**—When the convexity of the curve is felt in contact with the posterior pharyngeal wall, the point of the catheter is next *rotated inwards* through a quarter of a circle, *i.e.* through an angle of 90, as indicated by the metal ring, the instrument is now gently withdrawn until the concavity of the curve is brought into contact with the posterior free margin of the nasal septum.

**3rd Stage**—The stem of the catheter is now lightly grasped between the finger and thumb of the left hand just beyond the tip of the nose, so as to prevent it slipping, its point is then *rotated downwards and outwards* through half a circle and a little more, *i.e.* through rather more than an angle of 180, and thus it slips into the orifice of the Eustachian tube. At this stage the metal ring is directed outwards and upwards towards the external canthus of the eye of the same side. The instrument must be maintained in this position by grasping its stem

more firmly with the left hand Poltzer's bag is now inserted into the funnel-shaped outer extremity of the catheter and inflation is commenced. This must be done quietly and with the least possible jerking of the instrument, if the bag is provided with a valve it does not require to be removed from the catheter after each inflation. The air may be forced in as often as it is considered necessary. In order to remove the catheter from the Eustachian orifice the rotation is reversed, the point being carried downwards and inwards through a right angle. As the instrument is withdrawn from the nose, the hand must be carried down over the chin, so as to keep the point free from the posterior edge of the palate.

*Difficulties in the Use of the Catheter*—The elevation formed by the junction of the bony floor of the nose with the cartilagenous vestibule may prove an initial difficulty, unless the tip of the nose is tilted up in the manner already described. Spines or ridges growing from, or deviations of the septum, may obstruct the entrance of the catheter. When difficulties of this kind exist, they may be overcome by guiding the point of the instrument with the aid of the mirror and speculum, while in some cases it may be necessary to pass the catheter along the nasal fossa of the opposite side. Under these circumstances, stages 1 and 2 are the same as before, but instead of the point of the instrument being rotated downwards and outwards, as in stage 3, it is directed across the middle plane of the naso-pharynx by pressing the proximal end of the catheter outward against the ala of the nostril. It is better in such cases, however, to inflate the ear of the same side in the first instance, and then carry the point downwards and inwards across the middle plane to the corresponding spot opposite. Difficulties may be met with in the naso-pharynx: the curve of the catheter may be too pronounced to admit of proper rotation, so that one with a smaller curve must be substituted. Sometimes the movements of the soft palate interfere with the manipulation, when one or other of the following methods should be adopted in place of that already described. Stage 1 is the same as before, but instead of the point of the instrument being rotated upwards and then withdrawn until the concavity hitches against the septum as in stage 2, the instrument is withdrawn with its beak looking downwards until the concavity is arrested by the posterior edge of the hard palate, it is then rotated upwards and outwards through a quarter of a circle and a little more. On the other hand, some aurists prefer at the end of stage 1 to rotate the instrument at once upwards and outwards through a right angle, and then with the point against the outer wall of the naso-pharynx in Rosenmüller's fossa, they withdraw it, until it rides over the Eustachian cushion and slips into the orifice. This pro-

cedure may prove somewhat disagreeable to the patient.

*Information derived from Inflation*—If auscultation be simultaneously practised much useful information is obtained. When the air enters the tympanum through a patent Eustachian tube, the sound produced is full and clear, and the impact is apparently close to the observer's own ear. If the tube is obstructed the sound is fainter and appears more distant. If there is fluid in the tympanic cavity a faint moist sound may be detected, which must not be confounded with the louder gurgling noise produced at the pharyngeal opening of the tube. In the presence of a small dry perforation in the tympanic membrane the air may be heard whistling through it, sometimes, if the perforation is a large one, there is almost a painful sensation produced in the surgeon's ear. If the perforation is associated with secretion in the tympanum, a moist bubbling sound is heard. After inflation, the hearing power must be again carefully tested, and the result recorded.

*Complications resulting from Inflation*—In some cases giddiness and syncope have been produced as the result of inflation by Poltzer's method, while loss of consciousness accompanied by convulsions is a rare accident during catheterisation. Occasionally surgical emphysema has followed the use of the catheter, owing to abrasion of the mucous membrane by the point of the instrument. The greatest care should be practised in keeping the catheters clean so as to avoid the risk of any infection. The silver instruments should be boiled, while the vulcanite ones may be kept constantly immersed in a solution of carbolic acid without being damaged.

*Choice between Poltzer's Method and the Eustachian Catheter*—In the case of children the catheter is usually not employed. An objection may be expressed by adults to the use of this instrument, and, to obviate the discomfort which is frequently complained of by patients, some authorities recommend the inferior incus of the nose before its introduction. Poltzer's method, or one of its modifications, may in the first instance be used, unless there is any distinct contra-indication. The existence of a cicatrix or an atrophic condition of the tympanic membrane contra-indicates the use of this method, as the forcible introduction of air may cause rupture of the membrane in those areas. More accurate information can be obtained by the use of the catheter with regard to the degree of obstruction in the tubes, while the amount of air introduced can also be regulated. In other cases again in which there is marked obstruction, it is the only method by which air can be successfully inflated and the tube rendered more patent. As both tympana are simultaneously inflated by Poltzer's method, an unnecessary and even injurious strain may be thrown upon the healthy ear in cases where

repeated inflation is only required for one ear. Undue stretching of the structures in the normal tympanic cavity may to some extent be lessened by the patient inserting his finger into the external auditory meatus during the act.

**Ear, Local Anæsthetics**—To induce local anæsthesia for operations on the ear, the two drugs most commonly employed are *cocaine* and *eucaine*.

The anal operations for which local anæsthesia may be required may be grouped as follows—

**A On the External Ear**—Removal of cysts or small new growths from the pinna or outer part of the external meatus. Opening of furuncles. Curretting of granulations, etc.

**B On the Middle Ear**—Paracentesis of the membrana tympani. Removal of polyp. Curretting (Ossulectomy, simple opening of the mastoid, radical mastoid operation).

For the removal of cysts, etc., from the pinna or outer part of the external meatus eucaine may be used by injection. Local anæsthesia by freezing, by means of ether or other sprays, has been employed, but, owing to the structure and blood-supply of the auricle, is not to be recommended. Cocaine may be used by injection (in solutions of 5 per cent), but eucaine is probably the safer drug. Moreover, eucaine can be sterilised by boiling without undergoing decomposition, an advantage not possessed by cocaine.  $\beta$ -eucaine is soluble to the extent of 10 per cent, and, since its toxic effects are practically nil, there is no advantage in using any weaker solution. This solution should be injected by means of a sterilised hypodermic syringe into and beneath the skin at the site of tumours, etc., and ten to twenty minutes allowed to elapse before proceeding to operate.

For the opening of furuncles and the curretting of granulations, eucaine or cocaine may be used by instillation. This method is carried out as follows—The patient lies upon the sound side, with the ear to be anæsthetised uppermost. The solution of eucaine or cocaine, comfortably warmed, is then dropped into the meatus until that passage is full, and is retained for ten minutes. When the patient rises a pad of wool should be placed over the ear to absorb the superfluous fluid. This method may be used for the opening of furuncles, incisions into the meatus, the curretting of granulations from either meatus or tympanum, and for paracentesis of the membrane.

The above methods are, however, unsatisfactory at the best. The personal factor is so variable as regards the local anæsthesiation of skin surfaces that results ranging from good anæsthesia to nil will be met with. The intact *membrana tympani*, being covered with skin, in continuity with that of the meatus, is practically non-absorbent and, therefore, offers the greatest

obstacle to efficient local anæsthesia. Attempts to obtain the latter by means of agents which corrode the superficial epidermis have resulted in such mixtures as one of equal parts of cocaine hydrochloride, concentrated carbolic acid, and menthol. This method is, however, only relatively successful.

Shortly after this article was written for the first edition, Gray of Glasgow, taking advantage of the penetrating properties of aniline oil, introduced the solution known by his name "Gray's solution" consists of cocaine hydrochloride, 0.5, aniline oil, absolute alcohol, aa 50. But aniline oil being poisonous, instances of untoward results from its use have not been infrequent.

A better method than any of the above lies in the local application of dry crystals of cocaine on the end of a probe.

Since 1903, a modification of Schleich's method has been used in Politzer's Clinic at Vienna, and the workers there claim results so satisfactory as to justify the abandoning of general anæsthesia in a majority of cases. This method can be used for the removal of ossicles, and even for the radical mastoid operation. In this country, however, it will probably be long before otologists will operate in this manner by preference. It is stated that the hemorrhoidal attending operations by this method is so insignificant as to make it a distinct advantage.

Schleich's solution is a mixture of 1 per cent warm cocaine solution with five drops of tonogen (the trade name applied to Richter's extract of suprarenal gland). Tonogen corresponds to the "adrenalin" of Park, Davis and Co) to each cubic centimetre of cocaine solution. This mixture is injected under the periosteum by means of a special syringe. A full account of the technique to be carried out in operating upon the ear by this method is too long for insertion here; any abridgment would make the description inadequate. Two papers by Messrs Seymour Jones and Stoddart Barr will be found in *The Transactions of the Otological Society of the United Kingdom*, vol vi pp 127-136, in which the matter is dealt with clearly and fully.

#### External Ear, Diseases of. (Auricle and External Auditory Meatus)

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## ANATOMICAL AND PHYSIOLOGICAL OBSERVATIONS

—Under the name external ear are included the auricle and external auditory meatus. In form, in dimensions, and in the degree of angle of attachment to the head, the auricle or pinna presents great varieties in different persons and races of men. In structure, the auricle consists mainly of a framework of yellow elastic fibro-cartilage, lined by perichondrium which is covered by skin. The lower part or lobule, however, contains no cartilage, and is composed chiefly of skin, connective tissue, and fat. The skin lining the auricle is more loosely attached to the cartilage behind than in front, where sebaceous glands are found in great numbers. These muscles extend from the auricle to the side of the head—these are, no doubt, remnants of muscles originally intended to move the auricle, as a whole, forwards, upwards, and backwards. In few persons, however, are these muscles of more than very slight functional value. Traces of other six muscles, still less developed structurally and functionally, may be found connecting different parts of the auricle with one another.

*Functions of the Auricle*—The waves of sound falling on the concha are reflected forwards, and by the hollow formed by the tragus they are farther reflected into the meatus. It has been found experimentally that the artificial filling up of these hollows has appreciably weakened the hearing power in persons dull of hearing, who, on the other hand, often derive benefit by increasing with the hand the reflecting surface.

*External Auditory Meatus*—Extending from the auricle to the tympanic membrane is this somewhat winding passage, lined by a continuation of the cutaneous covering of the auricle. The width of the canal and the degree of its curvature present great varieties in different persons. Its length extends to about an inch, but, owing to the oblique position of the tympanic membrane at its inner end, the antero-inferior wall is longer than the postero-superior. The walls of the outer third of the meatus are cartilaginous, or rather membranous-cartilaginous, and are continuous with the auricle. It is to be noted that at birth there is really no bony meatus. The osseous canal develops gradually outwards from the squamous and mastoid portions of the temporal bone above and behind, while below and in front it develops from the annulus tympanicus, forming ultimately the tympanic plate. The tympanic membrane being almost horizontal at birth, the space between it and the floor of the meatus is then very narrow.

The lining of the meatus is skin, which gradually becomes thinner as it extends over the osseous portion, there it is firmly adherent to the periosteum, which really constitutes its deep layer. The lining of the cartilaginous meatus, especially at its inner part, contains the *glandulae ceruminosa*, which secrete the cerumen or ear wax, and are really modified sudoriparous glands.

*Nervous Distribution to the External Ear*—Sensory nerves are derived (1) from the cervical plexus, through the great auricular nerve, which supplies the greater part of the auricle, (2) from the third division of the fifth cranial nerve, through the auriculo-temporal, which gives branches to the outer surface of the auricle and to the walls of the meatus, (3) from the jugular ganglion of the pneumogastric the nerve of Arnold finds its way to the skin lining the posterior wall of the meatus. This nerve accounts for certain reflex phenomena, such as coughing, frequently observed when this part of the meatus is pressed upon with an instrument or foreign body. The muscles of the auricle are mainly supplied by the facial nerve through the posterior auricular and temporal branches. The otic ganglion is connected with the nerves which supply the various parts of the external and middle ear, controlling and regulating the reflex relations both to the various parts of the ear and to other regions of the body. From its connection also with the sympathetic, vasomotor impulses originated elsewhere may be transmitted through this ganglion to the lining of the external ear as well as other parts of the ear.

*Lymphatics of the External Ear*—The lymphatics from the auricle and meatus pass into the mastoid glands, the parotid glands, or the cervical under the meatus. Hence the glands in these situations are often found tender and swollen in connection with inflammatory affections of the external ear.

Diseases of the auricle and external auditory meatus, the parts of the organ of hearing having a cutaneous lining, come less frequently before the practitioner than diseases of the middle or mucous tract of the ear. Probably 20 per cent of the cases presenting themselves for treatment in connection with ear hospitals or clinics are affections of the auricle and external meatus. It is to be remembered, however, that middle ear diseases, especially the purulent variety, frequently involve the external meatus in one way or another. This is partly due to the anatomical relationship of the middle ear to the external meatus, and also to the fact that inflammatory products, formed in the middle ear, usually escape through the meatus, and in doing so may infect its lining membrane. It is, therefore, sometimes difficult to determine in a given case whether we have to deal with a primary affection of the meatus or one secondary to middle ear disease.

## I. DISEASES OF THE AURICLE

These will be described as follows — 1 Traumatic affections; 2 Malformations, 3. Cutaneous affections, 4 Morbid growths

1. *Traumatic Affections* — *Incised wounds* are common, involving, it may be, the complete severance of the auricle. In oriental countries this is frequently inflicted upon criminals and others. The lobule is sometimes lacerated, either gradually by the mere weight of an earring, or suddenly by the pulling of the earring, as is sometimes done by a child. The cleft thus produced may remain permanently

Simple incised wounds should be brought accurately together by sutures with antiseptic precautions, even when the auricle is almost completely cut or torn off careful suturing may lead to healing. If the auricle be entirely lost, an artificial one may be substituted. An old cleft in the lobule should be treated by carefully paring its edges, followed by suturing

*Traction and contusions* of the auricle may lead to serious consequences. The author has seen a severe form of mastoid periostitis excited in a boy through a man laying hold of the boy's ears and lifting him from the ground. Contusions or prolonged pressures may produce rupture of the cartilage with effusion of blood beneath the perichondrium, forming a swelling at the upper and anterior part of the auricle. This is traumatic *hematoma auris* or *otchermatoma*. The skin over the swelling may be unaltered, but more frequently it has a dark reddish-blue appearance, and there is usually a painful sensation of fulness. The swelling is sometimes due more to perichondritis caused by the injury, and the effusion may then be serous or sero-sanguineous, becoming in some cases purulent. Even when the effusion consists of blood, the colouring matter is apt to deposit itself on the walls of the cavity, and only straw-coloured fluid may escape on puncturing

The so-called *shunken ear* may result, especially if the cartilage has been torn, and is due to thickening of the tissues followed by contraction. It is occasionally seen in professional boxers, and is observed on some of the classical statues of athletes. *Hematoma auris* may, however, arise independently of injury, and the insane seem specially liable to this (see "*Hæmatoma Auris*")

*Treatment of Traumatic Hematoma Auris* — At first cold spirituous lotions, applied with moderate pressure, tend to reduce the swelling and to oppose inflammatory reaction. If the swelling continue, the application of blistering fluid will promote absorption. The author has found that aspiration of the fluid, with, in some cases, the subsequent injection of tincture of iodine, has been most successful. Failing these methods, especially if there be purulent formation, the swelling should be incised, the contents

removed, and the cavity treated by stuffing with iodoform gauze.

2. *Malformations of the Auricle*. — Certain malformations are connected with family and racial peculiarities, or with habits of dress. For example, the auricle may be seen lying unduly flat against the head, in elderly women, from the pressure of caps, so the prominently jutting out ear, occasionally observed, may be caused, or at least aggravated, by the habit of boys pulling their caps down between the auricle and the head. Heavy ear-rings are sometimes responsible for unsightly elongation of the lobule

The most important malformation of congenital origin is that of *defective development*. While entire absence of the auricle is very rare, it may be, on one or both sides, defective in form and size, or it may be represented simply by one or more small cutaneous or cartilaginous nodules or ridges. These rudimentary auricles may also be faulty in position, being frequently farther forward and lower down than normal, and the external meatus is usually either completely wanting, or is represented by a short canal, terminating in a *cul-de-sac*. The malformation usually involves some of the deep parts, especially those external to the labyrinth, including the osseous meatus, the tympano-Eustachian passage, and sometimes the Fallopian canal. In the latter case there is facial paralysis. Cases, however, have been reported, though rare, in which rudimentary auricles were associated with normal conditions of the other parts of the ear. As might be expected, from the nature of the process of development, the maxillary and palate bones are sometimes found also malformed, producing asymmetry of the face. In such abnormalities of the auricle and meatus there may be undoubtedly a certain amount of hearing, for it is to be remembered that the labyrinth, having quite a different mode of development from the middle and external ear, is not necessarily involved. While the labyrinth arises from an invagination of the primordial integument, the middle ear develops out of the first branchial cleft, from the posterior edge of which the auricle is formed

There is also the so-called *fistula auris congenita* — an indentation or closed canal occasionally met with in front of the tragus or on the helix, containing whitish material, and it is regarded as a remnant of the first branchial cleft

*Malformations of excess* are less important, and generally take the form of one or two cartilaginous or cutaneous nodules in front of a normal auricle — the auricular appendages of Virchow, regarded as very rudimentary additional auricles. Polyotia, or complete additional auricles, have been only very rarely met with

*Treatment* — For projecting ears a narrow, elongated portion of skin may be cut out, at the

junction of the auricle and the mastoid, and the wound carefully sutured. When the meatus is rudimentary, terminating in a *cul-de-sac*, an exploratory incision may be tried, but this is rarely attended by benefit, owing to the state of the deeper structures. For an æsthetic effect, if one auricle be rudimentary, an artificial auricle to match the normal one can be applied. This might, however, involve the operative removal of the rudimentary one.

3 *Cutaneous Affections.*—*Eczema* is the most common skin affection met with on the auricle. Certain applications, such as iodoform or linseed poultices, may be exciting causes, and the irritating effects of a discharge from the middle ear not infrequently account for the disease. In the acute state it may at first be mistaken for erysipelas. There are redness and swelling with heat and tension, soon vesicles appear, followed by the exudation of a yellowish fluid. The epidermis exfoliates, and, by the drying of the exudation, crusts form over the denuded cuts.

In the *chronic* form, however, *eczema* of the auricle is much more commonly met with, and it is then, in many cases, associated with a similar condition of the face and scalp. It is frequently confined to a part of the auricle. For example, in the form of a red moistened surface or fissure, covered with scales or crusts, we often meet with it at the junction of the auricle and the head, especially at the upper part. The fossa of the helix is also a favourite spot, and also the outer surface of the lobule and neighbourhood of the meatus, from the irritation of discharge from the ear. Fissures are apt to exude fluid and to bleed when touched. The skin of the auricle, in whole or in part, may ultimately become considerably infiltrated, and yellowish brown or reddish in colour, or it may be covered with inspissated secretion or epidemic scales. In certain neglected cases the auricle may ultimately become a thick brownish-red shapeless mass, fissured and covered with offensive crusts. The chief subjective feature, in the chronic stage, is itchiness, with a painful sense of heat and tension, when there is an acute exacerbation. There may be great induration and thickening of the skin at the outer part of the meatus, causing considerable loss of hearing.

*Treatment of Eczema of the Auricle.*—In the acute stage soothing applications only should be employed, while the auricle is at the same time protected from pressure. The parts affected should be gently but frequently sponged with a solution of subacetate of lead, in the proportion of one drachm to four ounces of water, while pieces of soft cloth dipped in the lotion should be kept constantly in contact with the morbid surface. In many cases dusting with a powder suits very well, such as—*R Pulv amyli* ʒij, *zinci oxid*i ʒi, *calaminæ* ʒss. *M*

Ointments, such as diachylon or benzoated

oxide of zinc ointment, are sometimes very useful, although occasionally ointments seem to do harm. The ointment should be kept constantly applied on a piece of soft rag. Hairs must be carefully removed from the neighbourhood, and the adjoining parts of the head, if these are affected with *eczema*, should also be treated. It is desirable, in severe cases, to protect the auricle with a light and soft covering, so as to avoid pressure or the contact of impurities. In the more chronic forms astringent and stimulating remedies are usually to be preferred. During an acute exacerbation, however, we must be careful to return to the soothing remedies. Mercurial and tarry preparations are indicated when there is chronic infiltration or great scabiness and dryness, with itchiness, but they should not be employed when there are moisture and heat. The following is a mercurial preparation of a milder form—*R Hydrag oxid rub*, *hydrag ammoniat* ʒā gr vj, *adipis benzoat* ʒj, *ol olivæ opt* ʒij. *M*

When a more stimulating remedy is demanded the following formula may be adopted—*R Liment calcis*, *ung hydri nitatis*, ʒā ʒi, *liq. carbonis detergent* ℥xij, *ung zinci oxid* ad ʒj. *M*

When crusts are difficult to remove, it may be necessary to saturate them with weak carbolic oil, or to apply a light warm poultice of bread crumbs and then use the milder stimulating preparations, such as any one of the following—benzoated oxide of zinc ointment, subnitrate or oleate of bismuth with benzoated lard or vaseline, diachylon ointment, or boracic acid and vaseline. The ointment should be smeared thickly on a narrow strip of soft linen which is laid into the various depressions of the auricle, it should rather be wiped off than washed with water—the latter being done as seldom as possible. The application of a solution of nitrate of silver (40 grains to the ounce of water) to chronic fissures is sometimes very useful.

*Internal remedies* are often of service. In the acute stage a saline aperient may be prescribed. In the chronic forms many cases are benefited by arsenic and iron.

Delicate tuberculous children should have cod-liver oil and iron with nutritious food, while the fresh open air of the country is, of course, desirable. In gouty subjects the use of Carlsbad salts, or preferably a visit to a spa, such as Harrogate in this country, or Carlsbad abroad, might be suggested.

Other affections of the skin are less frequently met with on the auricle, such as *herpes*, which here, as elsewhere, may be attended with severe neuralgic pains. *Lupus* occasionally affects the auricle both in the form of *lupus vulgaris* and *lupus erythematoses*.

*Erythema*, *seborrhœa*, *comedones*, are more frequently met with, but as there is no essential difference between these diseases, when they

affect the auricle, as compared with other parts of the body, the reader must be referred to them for further information.

*Erysipelas* frequently extends from the face or head to the auricle, and from the reddened, swollen, and vesicated appearance, may be mistaken for the acute stage of eczema. The author has seen *crispipelas* arise from the external meatus, and extend over the head during the course of a purulent middle ear affection.

*Abcess* of the auricle is generally of a furuncular nature, when it may be associated with a similar condition in the external meatus. *Abcess* may also arise from a traumatic cause. Warm fomentations, antiseptic applications, and incision are the remedies to be employed. The reader is referred to furunculus of the external meatus at p. 473.

*Frost-bite*.—When exposed to intense and prolonged cold, dermatitis of the auricle may be produced. The skin is red and inflamed, while in more severe forms, occurring particularly in persons having a feeble circulation, livid nodules, becoming pale on pressure, appear. These may lead to excoriation or ulceration, and, in the worst forms, to gangrene, which may result in extensive destruction of the auricle. In the early stage inflammation caused by frost-bite may be treated with iced water, or gentle friction, or the lead and opium lotion. The frequent bathing with hot water is said to be more efficient in some cases. The nodules are best treated with tincture of iodine or with collodion. Excoriation or ulceration should be treated by means of a suitable ointment, such as iodoform, or boric acid and vaseline. In persons predisposed to frost-bite, the auricle should be carefully protected from cold during severe weather, while the general health should be raised to as high a level as possible.

4. *Morbid Growths*.—Sarcoma and carcinoma rarely involve the auricle primarily. When originating in the auricle, epithelioma attacks most frequently the upper part of the helix, extending to the meatus, middle ear, and, as a rule, to the cranial cavity. Early extirpation of the disease by operation gives the only chance to the patient. Benignant connective-tissue formations, especially fibroma of the lobule, are most frequent. These fibrous growths of the lobule may originate in the irritation caused by the wearing of ear-rings, and they may attain to great dimensions, especially in negro women. Removal by operation is the only remedy. Angiomata are occasionally met with in the auricle. Several may exist, and they form bluish, pulsating tumours, extending, it may be, to the head. From them dangerous hæmorrhage may take place. Politzer recommends the thermopuncture by Paquelin's thermocautère. Sebaceous cysts, when occurring on the auricle, are treated as on other parts of the body. Gouty deposits, in the

form of sodium biurate, are sometimes met with, especially in the upper part of the helix or in the fossa of the helix.

## II. DISEASES OF THE EXTERNAL AUDITORY MEATUS

### 1. ABNORMAL STATES OF THE CERUMEN.—

*Excess—Ceruminous Obstruction*.—The ceruminous secretion consists, in a normal condition, chiefly of fatty and colouring matter forming a circular yellowish-white layer, having the consistence of honey, in the inner part of the cartilaginous meatus. When in excess it may be semi-fluid or doughy in consistence, but more frequently it is dry, firm, and even stony. The colour of the mass varies. It is often of a chocolate colour, while, if epidermis enters largely into its composition, the colour is grey or whitish. If the patient works in an atmosphere containing much coal-dust or other black particles, the colour may be a deep black. In addition to cerumen there enter into the masses sebaceous matter, epidermic scales, hair, coal-dust, etc., while, occasionally, the centre is composed of a small ball of cotton or other foreign body. The plug after removal may be found covered with a dirty white membrane, and, on its inner end, a mould of the outer surface of the tympanic membrane is occasionally seen. The obstructing mass not infrequently forms a plug filling the whole canal; but comparatively small quantities may, on the other hand, be sufficient, in certain circumstances, to impede the transmission of sound.

*Causes*.—These accumulations may be due simply to a mechanical hindrance to the natural escape of the cerumen from the ear, such as—a natural narrowness of the meatus, the presence of bristly hairs at the orifice, eczematous thickening of the meatal walls, exostosis or hyperostosis, or collapse in old age of the cartilaginous meatus. Efforts to remove the wax from the ear, such as by the use of the corner of a towel or an "aurilave," may result in the formation of a hard ball of cerumen in the deep part of the meatus. Or the obstruction may be simply due to excessive secretion, which in many cases is also abnormally dry and tenacious. This excess is often found naturally in persons who perspire readily; but local congestion, such as furuncular or eczematous inflammation, may excite the glands to abnormal action. Mechanical irritation also, such as frequent scratching with the point of a pen or a toothpick, may have a similar effect. Catarrhal processes in the middle ear are sometimes associated with excess as well as inspissation. In sclerosis of the middle ear, on the other hand, the secretion is often found to be diminished or even abolished.

*Symptoms*.—Defective hearing is the chief symptom of ceruminous obstruction. This may come on quite suddenly, and may for a time



fluctuate considerably, owing to changes in the size and position of the plug, effected by the entrance of moisture, sudden movement of the head, or efforts to allay itching. The accumulation may go on for a considerable time without causing deafness so long as there is an aperture, however slight, but the entrance of moisture during washing or in the bath may be the immediate cause of sudden and, to the patient, alarming deafness. Ultimately, the deafness becomes uniformly severe owing to complete impaction or hardening of the plug.

Subjective sounds, such as disagreeable buzzing, singing or rushing, are sometimes very annoying features of this form of deafness. They may be reflex from pressure on the nerve twigs in the meatus, or they may be due to direct pressure upon the tympanic membrane, and, through the ossicles, upon the labyrinthine fluid.

Pain in the ear is occasionally complained of, but a sense of itching or of "stopping up" is more commonly present. Pain of a severe nature is sometimes excited by inflammation of the lining of the meatus, due to the pressure of the hard mass.

Giddiness has been occasionally observed in connection with impacted cerumen.

The long-continued pressure of a ceruminous plug is capable of exciting inflammation of the tympanic membrane; in this way a perforation or opacity and thickening of the membrane may result.

*Prognosis.*—Although there is manifest plugging of the meatus we must not at once promise a cure, owing to the fact that there may coexist an incurable deeper-seated affection. In probably two-thirds of the cases we shall find that either complete recovery or improvement of the hearing will follow the effective syringing of the ear. If the deafness has come on suddenly, especially after such a cause as the entrance of water, and if there are marked fluctuations in the hearing, the removal of the mass will probably result in restoration. In many persons defective hearing from ceruminous collection tends to recur at intervals of months or years, and in such cases it proves, not infrequently, the precursor of other and more permanent forms of deafness.

*Diagnosis.*—The presence of excess of cerumen is easily demonstrated by the use of the speculum and reflecting mirror, when the external meatus may be found completely blocked, or the collection may be confined to the inner part of the canal, where it sometimes escapes detection by the unpractised examiner. Masses of epithelium, epidermis, or fungi; collections of dried pus coloured with cerumen (concealing, it may be, an old perforation), are apt to be mistaken for ceruminous collection. Careful examination may show that the meatus is not entirely closed by the mass, and that the deafness is really due to some other cause.

*Treatment of Ceruminous Obstruction.*—Syringing with warm water is the only safe and effective method of treatment. The syringing may at once be carried out if the mass be of a soft nature, but if hard and impacted, preliminary softening measures should be employed. The following solution is a suitable one:—℞ Sodii bicarb., aâdi carbolioi āā gr. vj., glycerini ʒij., aquæ ʒij. M.

After being warmed, half a teaspoonful is poured into the ear, and allowed to remain for ten minutes. This should be repeated twice during twenty-four hours before syringing. Owing to the swelling of the mass, by the absorption of the fluid, the deafness may, for the time, be aggravated, and the patient should be forewarned of this. In cases where we have to deal with masses composed chiefly of exfoliated epidermis or epithelium, there may be considerable difficulty in removing them owing to their adhesiveness; and the cautious and repeated use of forceps, followed by syringing, may be necessary before dislodging and completely removing them.

A brass piston syringe, capable of containing 4 ounces, is generally the most efficient instrument. The nozzle should have a smooth extremity, and should not exceed an inch and a half in length. The syringe should be furnished with a fixed ledge, or other contrivance, so as to prevent the slipping of the index and middle fingers during its use. A black vulcanite tray, with a concave edge to fit the irregular surface under the ear, is suitable for receiving the fluid as it issues from the ear; the concave edge should be pressed close to the skin, care being taken to prevent the escaping fluid passing down between the tray and the neck. Bubbles of air, mixed with the water, are very unpleasant to the patient, and are to be avoided by expelling the air from the syringe before beginning to syringe the ear. The water should have a pleasant warmth, not under 100° F. Cold liquid must on no account be used. During syringing, the point of the nozzle of the syringe is placed just within the external orifice, in contact with the roof, while the auricle is pulled backwards and upwards with the left hand. A fair amount of force is necessary; but, with interruptions, a stronger stream can be employed than is safe in cases of purulent disease of the ear. The medical attendant must not entrust the syringe to the patient, or to an incompetent person. In some persons, syringing the ear, even when done with care, excites giddiness, and occasionally considerable pain. These effects are more likely to be produced if the tympanic membrane be already perforated, especially if, at the same time, excessive force be used. When we have reason to suspect the existence of a perforation, we cannot be too careful in the use of the syringe, which, in such a case, must always be preceded by the antiseptic solvent already

mentioned. It is undesirable to continue the syringing after the meatus is quite clear, hence it is well to examine so as to ensure that the operation shall not be continued unnecessarily. On the other hand, we must make sure that the whole of the obstruction has been removed.

After the completion of the operation, any fluid which may remain in the ear is allowed to drain out, and the passage is carefully dried with absorbent cotton, it is well also that a plug of cotton-wool be worn in the meatus for some hours afterwards. Some injection of the vessels of the membrane and the inner part of the canal will be observed for a short time after syringing.

*Deficiency of Cerumen*—The canal of the ear is sometimes abnormally dry and destitute of cerumen, producing, it may be, a feeling of unpleasant dryness. This may coexist with defective hearing, which is, however, not a result of the lack of cerumen, but rather an *indication* of disease in the middle ear, probably of the sclerotic type. Treatment of the middle ear condition sometimes leads to increase of the ceruminous secretion. The sense of dryness may be alleviated by painting the walls of the meatus with vaseline or other lubricating ointment.

**2 FOREIGN BODIES IN THE EAR**—The foreign bodies most commonly found in the meatus are beads, stones of fruit, small buttons, peas, small stones, bits of bread, pieces of paper, fragments of wood or of slate pencil. They are most frequently found in the ears of children, and are usually introduced by the child, or a companion in play. When a child is brought in order that a foreign body be removed from the ear, we must make sure that it is really there, and must not rest satisfied unless we actually see it. For it not infrequently happens that, although a foreign body has been introduced into the ear, it has, unknown to the patient, found its way out again. The statement of the patient must therefore always be tested by the use of the reflecting mirror and the speculum. This is in most cases very easily done by any one having moderate experience in inspecting the ear. There may, however, in some cases, be difficulties in the way of actually seeing the foreign body. It may (1) have passed or been forced into the tympanic cavity, through a perforation in the membrane, (2) if a small object, it may be concealed in the depression at the inner end of the floor of the canal, especially if this be unusually deep, (3) it may be very difficult to see owing to the swollen condition of the walls of the meatus, (4) it may be embedded in cerumen, (5) a sharp object may have penetrated the skin of the meatus. In these various conditions, the careful examination by an experienced observer, with the help of a probe or syringe, will usually overcome the difficulty.

With regard to the symptoms produced by a foreign body in the ear, we must emphasise the fact that small, smooth, round bodies may re-

main for many years in the meatus without provoking irritation—if undisturbed. If there be but slight pressure on the walls of the meatus, the symptoms may be limited to a degree of dullness of hearing with sounding in the ear. On the other hand, if the object be larger, and especially if it has been forced deeply into the meatus, violent pain may be excited, both from the pressure and from the inflammation which ensues. Rare cases are recorded of persistent cough, constant sneezing, giddiness, and vomiting, and even epileptiform attacks, due to foreign bodies in the ear, the symptoms disappearing after their removal. There is no doubt, however, that the most serious consequences of foreign bodies in the ear have been due to the injudicious or unskilful use of instruments for their removal. The author has known death itself result from such efforts, and he has also known grave injuries inflicted on the structures of the ear from the use of instruments intended to remove a foreign body, which was, however, proved to have no existence there.

*Removal of Foreign Bodies from the Ear*—If the foreign body be smaller than the lumen of the canal, which it usually is, and if it has not been driven in beyond the narrow part of the meatus (the isthmus), the use of a syringe and warm water will, in the majority of cases, ensure the expulsion of the body. If we find on examination a free space between the walls of the meatus and the foreign body, the stream of warm water should be directed into it. A syringe such as that used for the removal of impacted cerumen is suitable, and the amule should be pulled well upwards and backwards with the left hand, while the child's head is inclined somewhat downwards towards the affected side, in order to assist the movement of the object outwards. When the foreign body has reached the outer orifice, it can be easily removed with a small scoop. It is to be noted that, if the foreign body be a pea or other vegetable substance, it may, from absorption of the fluid, become more firmly impacted after syringing. We should therefore be prepared to use other means immediately after the failure of syringing.

When, however, the foreign body is found to be impacted deeply in the canal, especially if it has been forced, either at its introduction or in the subsequent efforts to remove it, beyond the narrower portion of the canal, into the space where the floor dips down and the canal widens, its removal may be a matter of great difficulty. If, through inflammatory swelling of the walls of the meatus, the foreign body has become immovably fixed, the difficulty may, for the time being, appear insuperable. In such tightly impacted foreign bodies we should first examine carefully for any space, however small, between the foreign body and the walls of the meatus, so that a stream of water from a syringe may be directed into the space and behind the foreign

body, with the hope that the latter may be urged, by the pressure of the fluid, towards the external orifice. If, after a fair trial, syringing should fail to expel the object, some form of instrument must be resorted to. No one, however, should attempt extraction with an instrument in such circumstances, especially in the case of a child, without the use of chloroform or other general anæsthetic.

The patient being under the anæsthetic, the interior of the ear is thoroughly illuminated, through a speculum, by means of a reflecting mirror on the forehead. A thin but strong instrument, slightly curved, such as one of the blades of Dr. Guy's forceps (miniature midwifery forceps), is introduced between the foreign body and the wall of the meatus, selecting a gap, if such exists, and used as a lever to urge the body outwards. Such an instrument should be as fine and small as possible, compatible with sufficient strength, because it is often a matter of much difficulty in such cases to pass even a thin instrument between the walls of the meatus and the foreign body, while the force which may be necessary in order to prise the object outwards can only be safely employed with a strong instrument. If practicable, the instrument should be introduced antero-inferiorly, where the tympanic membrane is farthest away from the orifice of the ear. Lister's hook is suitable when the foreign body is not too deeply situated. A convenient instrument is to be had having Guy's curved arrangement at one end and Lister's hook at the other. Ordinary forceps should, as a rule, be discarded, as they tend to impel the object farther in, only when the foreign body is close to the orifice of the ear, or very small in size, are they permissible, and then syringing is preferable. If we are able to encircle the object with a strong wire loop, used with Wilde's snare, it may be possible to bring it safely away, or so change its position that syringing may be successful, or the use of the lever instrument rendered possible. In the case of a vegetable substance, such as a pea, tightly impacted, a fine bent hook may be used to break it down, afterwards the fragments are expelled with a syringe. Lowenbergs suggested that a fine brush, dipped in a solution of glue, should be applied to the foreign body, with the hope that adhesion may take place and allow of the foreign body being safely withdrawn. In the case of long-pointed objects, lying across the meatus, it may be necessary to break them with forceps and extract the pieces separately or syringe them out. If the foreign body has passed through the tympanic membrane into the tympanic cavity, its removal with the wire loop should be attempted. The author has not found any help in such cases from the air douche, or fluid injections through the Eustachian tube, or suction with Siegle's speculum, as suggested by some authorities.

If these various methods prove unsuccessful—the foreign body being immovably wedged in the inner end of the meatus or in the tympanum—we should make a long incision at the junction of the auricle with the mastoid process, dissect the auricle and cartilaginous meatus forwards, and thus gain access to the osseous part of the meatus, nearer to the foreign body. A strong lever instrument can then be more safely and effectively employed. The wound should be carefully sutured, and the meatus plugged with iodoform gauze for a few days. In the event of failure, as a last resort the posterior wall of the osseous meatus should be removed with the bur or chisel, and, if necessary, the autium mastoideum can be opened.

Insects, such as fleas, earwigs, bugs, etc., may find their way into the ear and excite alarm in the mind of the patient. Their presence may not only produce the sensation of a moving body in the ear, but, by fastening upon the skin of the meatus or the tympanic membrane, severe pain may be excited. There is no evidence that the earwig is an especially dangerous inmate of the ear, as popularly believed. Maggots may be found in the ear, especially if the latter be the seat of a purulent discharge, from the deposition of the eggs of the fly, probably attracted by the purulent odour. Insects are easily expelled from the ear by syringing with water, or even by pouring water or oil into the ear, allowing it to remain for a few minutes. The smoke of tobacco, blown into the meatus, usually leads to the departure of the insect. In the case of maggots rectified spirit may be poured into the ear, and after a time the use of the syringe will generally expel them. It may be necessary, however, to pick them out with forceps. If a leech should find its way into the ear, syringing with a solution of common salt will ensure its expulsion.

**3 FURUNCLE OR CIRCUMSCRIBED INFLAMMATION OF THE EXTERNAL MEATUS**—Synonyms *Otitis externa circumscripta*, boils in the ear. This is one of the commonest inflammatory affections of the external meatus, and is often associated with boils elsewhere. The inflammation has its origin in a hair follicle or gland in the subcutaneous tissue, generally in the cartilaginous part of the meatus. A core of sloughed tissue usually results with more or less purulent formation, the latter may be very slight, or it may constitute a distinct abscess. We generally find more than one in the same ear, and they have a marked tendency to recur.

**Causes**—While no doubt essentially microbic in origin, local irritating conditions are important factors in the causation of these furuncles. The special microbe, which enters the hair follicle and excites the inflammatory process, is the *staphylococcus pyogenes*, aureus or albus. The disease is often associated with scaly eczema of

the meatus, when the mechanical efforts to relieve the feeling of itchiness seem to induce it. Furunculi are also a not uncommon complication of purulent middle ear disease, probably owing to infection derived from the purulent discharge. The constant presence of moisture in the meatus, the temporary entrance of cold water while bathing, or the use of irritants by way of treatment may excite the disease. Like most diseases, it is often attributed by patients to cold. No doubt certain defective states of the health may predispose to these furunculi, such as diabetes (See "Boils").

*Symptoms.*—Pain in the ear is the chief symptom. This is sometimes intensely acute, radiating, it may be, over the side of the head, while frequently there is a painful pulsation in the ear described as a "hammering"; these painful sensations are always worse at night. Movements of the auricle tend to aggravate the pain, so does pressure, such as lying upon the auricle, or pressure on the tragus. The pain is also intensified by movements of the lower jaw, as in chewing, yawning, and even speaking. When the boil is on the posterior wall of the meatus there may be pain on pressure over the mastoid, which may sometimes be oedematous, while, if over the anterior wall, the front of the tragus may be swollen and very tender to touch. The hearing is usually for the time defective, owing to the partial or complete occlusion of the meatus by the swelling, while there is often a humming or buzzing sensation in the ear. In severe cases febrile disturbance may be present. When examining the ear we should at first simply reflect light into the meatus without a speculum, as the use of the latter may be very painful. If there be any secretion in the canal the latter should be gently syringed and carefully dried with cotton-wool. We may then find two or even three furunculi in different stages, and the meatus entirely closed by the swelling. The skin over the furunculus is usually reddish, and when pressed with a probe is extremely sensitive.

*Course.*—The inflammatory centre may develop into a distinct abscess, but more frequently, after a few days, the boil ruptures, giving exit to a small slough and a few drops of pus, with relief to the pain. The pus or slough may require to be pressed out of the little orifice with a probe. Granulation tissue occasionally sprouts from the opening, but the disease very rarely leads to any affection of the bone. Excessive formation of epidermis or cerumen sometimes follows an attack.

*Diagnosis.*—The nature of the affection is determined by the soft localised swelling, very sensitive to pressure with a probe, and also by the pain elicited during pressure upon or traction of the auricle. It may in a slight case look like a small pimple at the orifice of the ear. We must not confound the oedema over the mastoid with actual periostitis.

*Treatment.*—Antiseptic and sedative plugs introduced and kept in the ear are very useful, such as an ointment composed of 1 grain of menthol, 2 grains of iodoform or boracic acid, and 1 drachm of vaseline, this is smeared thickly on long cotton-wool plugs, introduced well into the meatus and changed every few hours. Carbolic acid and vaseline, 1 in 50, may be used in the same manner. An ointment composed of 1 gram of hydrochlorate of morphine to 1 drachm of vaseline has a sedative effect. These plugs should not be allowed to press painfully on the walls of the meatus, but made simply to occupy the canal so as to bring the antiseptics in contact with the inflamed areas. Some prefer the use of antiseptic solutions, especially in the recurrent form of the disease, such as boracic acid and alcohol 1 in 20, or perchloride of mercury 1 in 2000, but the author has found the antiseptic plugs preferable.

Warmth and moisture, in the form of linseed-meal poultices or hot fomentations, frequently applied over the ear, into which an antiseptic plug has been placed, are distinctly useful in relieving the pain. These applications should, however, be discontinued as soon as the pain is relieved, as there is no doubt that excessive poulticing tends very much to the recurrence of furunculi. As a rule it is well to avoid the entrance of liquids, such as oils, into the ear, and we should aim rather at keeping the ear perfectly dry, with the exception of the antiseptic plugs. If syringing be necessary, the ear should be afterwards most carefully mopped out with absorbent cotton-wool. After syringing and drying, the insufflation of a small quantity of fine boracic powder, before the introduction of the plug, is useful where the canal is not completely closed. If the furunculi affect an ear already the seat of purulent middle ear disease, it is doubly important to employ dry treatment. In such a case the ear as a rule requires to be syringed with antiseptic solutions, but great care should be taken to dry the whole of the meatus by means of absorbent cotton, on a cotton-holder. A little dry boracic powder is then blown in and the antiseptic cotton-wool plug worn.

If the pain, in spite of such remedies, continues so as to prevent sleep and cause much suffering, an incision is made into the seat of the inflammation. A slender and sharp-pointed blade, such as a fine tenotomy knife, is suitable, and we should cut from below upwards, this being less painful. The contents of the boil are then pressed out with a probe, and the foregoing treatment carried out. If an anæsthetic be not administered, the head should be held firmly, as the inflamed tissue is very sensitive to incision, which, however, is generally soon afterwards followed by marked relief. Granulation tissue, if present, should be removed with forceps followed by the use of boric powder.

If there be chronic scaly eczema of the meatus, appropriate treatment must be employed, so as to prevent recurrence of the boils.

In persistently recurrent furunculæ, general treatment may be very useful. We should regulate the diet, the use of stimulants, exercise, etc. The possibility of the patient suffering from diabetes should be kept in view. If there be anemia on the one hand, or plethora on the other, we must prescribe appropriate treatment. Arsenic may be employed in the persistent form, while sulphide of calcium is a remedy worthy of trial.

4. **DIFFUSED INFLAMMATION OF THE EXTERNAL MEATUS—*Otitis Externa Diffusa***—This includes a variety of conditions in which the cutaneous lining of the meatus, often including that of the tympanic membrane, is involved in the inflammatory process. In the severe forms the periosteum of the bony meatus usually participates.

The following varieties may be distinguished.

(a) *The acutiform form*, which manifests itself in the acute stage by redness and swelling, with copious serous exudation and epidermic formation. It is frequently based upon chronic scaly eczema of the meatus, or it may be an extension from eczema of the auricle. Iodoform, used in the treatment of middle ear disease, is sometimes responsible for it. Chronic eczema may bring about stenosis of the meatus from gradual thickening of its lining membrane.

(b) *Traumatic Otitis Externa*—This form may be due to (1) the insertion of foreign bodies in the ear, or, more commonly, improper attempts to remove them, (2) the introduction of irritating or caustic substances, such as sealding water, cold water, chemical and caustic substances, (3) the unskilful use of instruments, (4) injuries, such as punctured wounds, fracture through the roof of the meatus, fracture of the tympanic plate. The latter may be caused by a fall from a height in which great force is applied to the chin, the bleeding from the ear which takes place in this injury may be readily mistaken for fracture of the base of the skull.

(c) *Syphilitic Otitis Externa*—This is a rare affection, and may occur either in the primary or in the secondary stage of the disease. It is usually seen in the form of condylomata and ulcerations, which are apt to leave cicatrices or pigmentary patches, the former sometimes seriously contracting the canal. They usually occupy the outer orifice of the ear, appearing as a greyish-red mass of granulation tissue, yielding a fetid discharge. The ulcerations have a dirty-white appearance, circular in shape.

(d) *Parasitic Otitis Externa*—This form (otomycosis) is due to the growth and accumulation of fungi, generally belonging to the genus *aspergillus*, found chiefly on the inner third of the canal, and the outer surface of the membrane. Damp dwellings generally favour

their formation. The fungi seem to develop most frequently in a meatus where there has been an accumulation of epidermis or cerumen, forming a nidus for the germination and growth of the parasite. The two chief forms of fungi found in the ear are, 1st, *aspergillus nigriscans*, forming a blackish collection, 2nd, *aspergillus flavescens*, in which the spores form yellowish points—the collections being whitish or greyish. These fungi adhere very tenaciously to the osseous walls of the canal and to the tympanic membrane, and, when removed, the cutis beneath is red, presenting sometimes a bleeding tendency.

If the fungi be not eradicated there is a great tendency to relapse of the inflammation with much pain. No doubt these collections of fungi are sometimes regarded as simple ceruminous or epidermic accumulations. If these show a tendency readily to recur, and especially if there be considerable pain, microscopic examination should be made.

(e) *Croupous and Diphtheritic Inflammation*—This form is rare, and is generally associated with pharyngeal diphtheria, although it has been known to occur in the meatus independently of the throat. In this variety there is a dirty greyish membrane, which adheres to the osseous meatus, and on the separation of the membrane a bleeding surface is exposed.

(f) *Secondary Otitis Externa*—In acute otitis media the lining of the osseous meatus is almost always involved, but the chronic middle ear purulent affections still more frequently involve the meatus. The mastoid antrum and mastoid cells are closely related to the postero-superior wall of the bony meatus, so that purulent collections not infrequently make their way through the layer of bone separating these spaces and burrow underneath the lining of the meatus, or, bursting through the lining, a fistulous communication with the meatus is formed. The greater part of the postero-superior bony wall may thus be destroyed, with or without the formation of sequestra. Even the whole of the bony tissue intervening between the meatus and the anterior knee of the lateral sinus may in some cases be destroyed. These conditions show themselves by profuse discharge and by exuberant granulation tissue or polypi which spring up and frequently fill the meatus.

*Symptoms of these various forms of Otitis Externa*—In the acute stage the subjective symptoms consist of pain, impairment of hearing and subjective sounds, they can scarcely be distinguished from those attending the circumscribed variety of inflammation. Like the latter the pain is usually aggravated by movements of the jaw, or by pressure or traction upon the auricle, and the hearing is impaired in proportion to the degree of swelling, and the amount of inflammatory products, as well as to the degree in

which the tympanum participates. In severe forms, at a very early stage, the epidermis may be elevated by effused blood, causing one or more bluish swellings in the bony meatus, which may lead to considerable discharge of blood from the ear (*otitis externa hemorrhagica*). After a short period of hyperemia and swelling, with, it may be, hemorrhagic elevations, a discharge from the meatus appears. In the eczematous variety there are usually itchininess, heat, and sense of fullness, and, on examination, we find in the meatus serous or purulent secretion, with, in many cases, laminated masses of sodden epidermis. After removing inflammatory products by cautious syringing and drying, the meatal and tympanic surfaces are usually seen to be red, swollen, spongy, or granular from loss of epidermis. In the chronic stage, granulations are apt to spring up, while the discharge acquires a disagreeable odour. Glandular enlargements of the neck or swelling over the parotid gland are likewise not uncommon. If treatment be neglected or insufficient, especially if the patient have an unhealthy constitution, the disease is apt to go on indefinitely, and may bring about such consequences as the following—(1) stenosis of the meatus, from hypertrophy of the cutaneous lining, or from hyperostosis of the bony walls, (2) caries or necrosis of the osseous part (this is, however, a much more frequent result of middle ear disease), (3) perforation of the tympanic membrane and extension to the middle ear, or opacity and thickening of the tympanic membrane, (4) owing to the defective state of the tympanic plate in the young child and to the clefts in the cartilaginous meatus, inflammation of the external meatus is apt, at that time of life, to extend to the articulation of the jaw and the parotid gland, (5) fatal implication of the meninges, brain, or lateral sinus may result by extension through the roof or back wall. This, however, is very rare compared with fatal extension from the middle ear spaces.

As inflammatory affections of the meatus are very often associated with middle ear disease, it is sometimes difficult, in a given case, to decide which is the primary condition, while, owing to stenosis of the meatus, it may be impossible to determine accurately the state of the tympanic membrane or middle ear. From funicular inflammation the diffused form is distinguished by the localised nature of the former as tested by a probe. If the inflammation be eczematous, we shall find other symptoms of eczema, such as itchininess, scaliness, and occasional serous discharge. In the parasitic form, microscopic examination is necessary in order to form a correct diagnosis. This variety is to be suspected, if a condition resembling a ceruminous collection is attended by severe pain, difficulty in clearing out the meatus, and tendency to recur.

*Treatment of the various forms of Otitis Externa Diffusa.*—In the acute stages, abstraction of blood, by means of leeches, will have a mitigating influence, especially in removing pain. Two leeches applied over the tragus, and two at the lower part of the mastoid, will afford sufficient depletion in the case of an adult. After the bleeding has ceased, the good effects are enhanced by the use of hot fomentations. In milder cases, or when there is not much pain, removal of blood is unnecessary. Gentle syringing with hot water, in which 2 per cent of boracic acid has been dissolved, is also calculated to soothe the pain. Warm poultices of linseed meal applied over the ear are also soothing, but they should only be used while the pain is at its height, and should be discontinued as soon as there is relief. A few drops of tincture of opium or of the following—*R* Liniment belladon., Iminut. opii ʒi ʒiv M—placed on a plug of cotton-wool, and inserted in the outer orifice, usually afford relief to pain. The gelatine preparations of Gubler (*amygdalus aurium*) are preferred by some. These contain, for an adult, either  $\frac{1}{4}$ th of a grain of liquid extract of opium, or  $\frac{1}{4}$ th of a grain of hydrochlorate of morphine. They are introduced with aural forceps, and the meatus is afterwards closed with cotton-wool. The gelatine gradually dissolves, and relief to the pain follows. If the pain should be intense, preventing sleep, an internal sedative, such as Dover's powder, or the subcutaneous injection of morphine, may be required at night. In the acute condition rest and quiet in the house, especially during winter weather, contribute to the recovery. At the same time a plug of cotton-wool should be kept in the orifice of the ear, while a pad of cotton-wool is placed over the side of the head.

When the secretory stage begins and inflammatory products occupy the meatus, the ear should be gently syringed once or twice a day, or less frequently if the secretion is slight, with a hot solution of boracic acid, then dried as far as the tympanic membrane with absorbent cotton on a cotton holder, and a small quantity of finely powdered boracic acid blown in. As soon, however, as the secretion has ceased to form, the parts should simply be kept dry. Granulation tissue must, if present, be removed with a snare or suitable forceps, followed either by the boracic treatment, just described, or by the spirit treatment. The latter form of treatment is applied as follows—After syringing with a solution of boracic acid and drying out the ear, a solution, consisting of equal parts of rectified spirit and water, well warmed, should be poured into the ear and allowed to remain there for ten minutes—repeating the process twice a day till the ear be perfectly dry. When the condition of the meatus depends upon a purulent affection of the middle ear, these remedies are specially suitable.

In the eczematous form of inflammation, when the walls of the canal are thickened, and in the dry scaly condition, elongated plugs of cotton smeared with an ointment such as the following—Hydrarg. oxid. rub., hydrarg. ammoniat. 33 gr vj, adipis benzoat. 3j, ol. olivæ opt. ʒij M—should be inserted twice a day, so as not only to stimulate absorption, but also to exercise a certain degree of pressure. In the more persistent forms of thickening, a strong solution of nitrate of silver (40 grains to the ounce of water) should be painted over the walls of the meatus twice a week, for several weeks. After each application a ball of cotton, soaked in a solution of common salt, is applied for a few seconds to the orifice of the ear to prevent blackening of the skin. In the dry scaly form, without stenosis but with troublesome itching, we should use, in addition to the foregoing treatment, the following—R Acid. carbolicæ gr v, spirit. rectif. ʒij, glycerum ʒij M Sig.—For painting external meatus twice daily.

If the inflammation be of a specific nature, general anti-syphilitic treatment should be employed. The condylomata may be cut off with scissors, and chromic acid then applied. Occasional dusting with calomel powder will also prove useful. An ointment, containing 10 grains of iodoform to an ounce of vaseline, may sometimes be applied with advantage.

In the treatment of the parasitic form, the effective use of the syringe, with a 2 per cent solution of boracic or carbolic acid, as already mentioned, may have to be repeated several times before the masses can be removed, and the aid of forceps may be necessary. Some decided parasiticide, such as rectified spirit or an alcoholic solution of bichloride of mercury (1 grain to the ounce of rectified spirit), twice a day for a week, may be required to prevent recurrence. Such solutions, after being poured into the ear, should be allowed to remain there for five minutes.

While local treatment is of first importance in these inflammations of the external meatus, general treatment, especially in the chronic forms, contributes to recovery, such as the use of iron, cod-liver oil, nutritious food, abundance of fresh open air, etc.; anti-syphilitic remedies in specific cases, arsenic in eczematous cases, alkalies and aperients in gouty conditions.

**5. CARIES AND NECROSIS OF THE EXTERNAL MEATUS.**—While caries and necrosis may occur primarily from inflammation of the osseous part of the meatus, it is important to remember that bony affections of the meatus are usually part of a purulent middle ear disease, especially affecting the posterior wall through the proximity of the mastoid antrum and cells. In such cases the upper and back wall may be first seen to bulge, owing to pus forming under the skin and periosteum. The bone forming the outer wall of the attic of the tympanum is not infrequently involved, leading to exposure of the

head and neck of the malleus. In caries and necrosis there is a purulent, often sanious, discharge from the ear, and exuberant granulations spring from the seat of the diseased bone, which bleed readily when touched. These may fill the canal of the ear, and they recur after removal. There are often other manifest evidences of mastoid and tympanic disease. A probe may show bare firm bone, especially on the back wall, at other times there is a movable sequestrum or the probe may pass through a carious aperture into the cells behind.

*Treatment of Caries and Necrosis.*—A soft bulging in the upper and back part of the canal may first require to be incised, when caries or necrosis of the bone beneath may be found. In the case of a movable sequestrum, syringing may be sufficient to remove it, failing that, a pair of strong forceps must be employed, while the patient is under chloroform. A long-pointed sequestrum, fixed transversely in the meatus, may require to be broken before removal. Sequestra of considerable size, usually coming from the mastoid wall, are often removed from the meatus. One or more necrotic ossicles may also escape, or be removed from the meatus. Care must be taken, after the removal of a sequestrum, to prevent stenosis, by plugging with narrow strips of iodoform gauze. In many cases caries or necrosis of the postero-superior wall of the meatus can only be dealt with efficiently by operating through the mastoid. It may then be found that the postero-superior wall of the meatus has been already destroyed by caries or necrosis, or the operative treatment may necessitate the removal of that part of the meatus. Where the middle ear spaces are really the sources of the mischief, the thorough treatment of the purulent middle ear disease must be resorted to. This will be found described in the article on purulent disease of the middle ear. A superficial carious spot can be safely and successfully curetted by a small sharp spoon, and the operation should be followed by the application of iodoform and boracic acid, one of the former to three of the latter. Any constitutional defect or cachexia must be treated by appropriate remedies.

**6. STENOSIS OF THE MEATUS.**—(a) *Stenosis from Malformation.*—The orifice of the ear may be reduced to a mere slit, from the approximation of the anterior to the posterior wall, due to an over-lapsed state of the fibrous tissue and skin in old persons. If this interferes with hearing, a small silver tube, having the shape of an ear speculum, may be kept in the ear during the waking hours. Congenital malformation is, however, more important, when it is usually found in connection with a congenital defect of the auricle, the meatus being at the same time absent or represented by an indentation, or a small canal terminating in a *cul-de-sac*. There are also usually in such cases congenital defects

in the middle ear, and, therefore, operative efforts to form a proper canal very rarely result in benefit to the hearing. In a very few cases, where the congenital contraction has been limited to the outer end of the meatus, benefit has followed the cautious use of spongo tents. A partial closure of the meatus is sometimes caused by a marked projection of the antero-inferior wall of the osseous portion—this only proves inconvenient by impeding the examination of the lower part of the membrane.

(b) *Stenosis from Swelling or Thickening of the Cutaneous Lining*.—The most common cause of this form of stenosis is chronic eczematous inflammation. The persistent irritation of the meatal lining, caused by a purulent discharge from the middle ear, may also in time lead to swelling and thickening, causing partial stenosis. Under this heading may also be included partial or complete stenosis due to abscess, tumour, or enlargement of the parotid gland in front. Pus may, in the former case, find its way from the parotid into the meatus. Furunculi may also completely close the meatus. Swellings arising from mastoid disease frequently also obstruct the meatus from behind.

In the treatment of these forms of stricture of the meatus, we should first clear away, by syringing with a warm solution of boracic acid, any materials, such as purulent, ceruminous, or epidermic collections, which are apt to collect in a narrow channel. If there be eczematous thickening and no middle ear disease, we should treat this as already described. In many cases, however, we have to deal with a discharge from the middle ear. Then careful cleansing and drying of the narrow passage, along with gentle efforts to dilate it, should be carried out. The more regular introduction of a cotton-wool cylinder, for the purpose of drying the passage, has a somewhat widening effect, and still more so if the elongated plug be smeared with some stimulating ointment, such as iodoform or boracic acid and vaseline, and retained constantly in the ear, changing it night and morning. Such plugs should be gradually increased in thickness, as the increasing lumen of the canal permits. Patients, when taught to introduce these simple plugs, can often effect marked improvement, even when the thickening is partly bony in character. With increased width of the meatus, treatment of the middle ear can be more efficiently carried out. In the more intractable forms, sponge or lamina tents, preferably the former, may be tried, but with caution. A severe and rapid dilatation must be avoided, and the tent should not be allowed to remain in the ear more than an hour at a time, nor repeated more frequently than every third or fourth day, while the surgeon should be at hand to remove the tent if much pain is excited. A small conically shaped vulcanite or rubber tube, placed and retained in the canal after the dilating

efforts have ceased, may be useful in maintaining the dilatation, while it facilitates syringing as well as the escape of secretion from the middle ear.

(c) *Stenosis from the Formation of Septa or Adhesions*.—A membranous septum sometimes forms across the meatus, leading to its entire occlusion. It may be mistaken by the inexperienced observer for the tympanic membrane, but its nearness to the outer orifice and the absence of the usual features of the tympanic membrane distinguish the one from the other. The septum more frequently takes the form of a membrane with a hole in the centre. A certain extent of the meatus may be closed by adhesions, due to the long-continued contact of ulcerated surfaces, or to the coalescence of granulation tissue. The defective hearing in such cases will depend on the extent of the closure and on the condition of the middle ear. Where we find septa or adhesions, the middle ear is or has been the seat of purulent disease.

A membranous septum, closing the canal, should be removed by a circular incision, and an antiseptic cotton-wool plug or a strip of iodoform gauze introduced, so as to ensure an open space, until the healing process has been completed. Adhesions have to be treated by separation with a fine-bladed knife, followed by plugs, as in the case of septa.

(d) *Stenosis from Hyperostosis of the Meatus*.—Hyperostosis is frequently due to chronic purulent middle ear disease, giving rise to chronic periostitis of the meatus with increased formation of bone. The cutaneous lining is also usually reddish and somewhat thickened. The lumen of the canal may be reduced so as scarcely to admit of an ordinary probe, and, when due to purulent middle ear disease, usually contains secretion. It is to be remembered that if the closure of the meatus be very great in these purulent conditions of the middle ear, dangerous retention of pus in the deep parts may result. Hyperostosis is occasionally found in connection with non-purulent middle ear catarrh. From exostosis, hyperostosis is distinguished by the diffused uniform thickening of the latter.

The treatment of this form of stenosis is included in that of the second variety.

(e) *Stenosis from Exostoses or Osseous Tumours*.—There are two varieties of exostoses of the meatus—(1) those with broad bases, usually multiple, very hard, even ivory in texture, and terminating in apices, which approach each other so as to leave a small space between; (2) those having a narrow pedunculated attachment, usually springing from the posterior meatal wall, and occurring singly.

*Cause*.—Aural exostosis seems frequently to be associated with hereditary predisposition or constitutional peculiarity, and the ivory or multiple varieties are probably in many cases connected with the gouty or rheumatic diathesis. They are certainly found more commonly in



men of middle age who eat and drink generously. The ivory varieties are not only usually multiple in the one ear, but they are as a rule found in both ears. The softer or pedunculated variety has, no doubt, in most cases, its origin in irritation or inflammation of the posterior wall of the meatus, secondary, in most cases, to an inflammatory process in the mastoid cells, which has led to periosteal thickening or a small abscess. It is supposed that granulation tissue first forms, which gradually undergoes a process of ossification.

It seems probable that the frequent entrance of cold water into the ear tends to lead to these growths, and we often find on inquiry that patients with aurial exostoses have been much in the habit of diving in water during the earlier periods of their lives. It has been found by Blake and others that the crania of the aboriginal inhabitants of America, who lived on river banks, show the presence of exostoses in the ears remarkably frequently, the explanation being that they spent much of their time in the water. Aurial exostoses are also said to be common amongst the South Sea Islanders, who are notable divers in the sea.

**Symptoms and Course.**—Aurial exostoses are frequently found in persons who are not conscious of anything being wrong with the ear. The patient may come to the surgeon owing to deafness due to some other cause, when the presence of these growths is incidentally found by the surgeon. In these cases the surgeon may see several knob-like projections, of ivory hardness, pale in colour, very sensitive to the touch of a probe, and existing in both ears. There is usually a space between their apices, through which a portion of the tympanic membrane may be seen. This space may, however, at some time or other become closed by epidermic or ceruminous collections, causing deafness, which brings the patient for advice. The pedunculated variety, springing from the bony back wall near its junction with the cartilaginous, is more likely to go on to complete closure of the meatus, the skin covering it becoming slightly red and thickened. When complete closure takes place pain may be excited, and there is serious impairment of hearing. It is self-evident that, when the meatus is quite blocked by the growth, the occurrence of a purulent discharge in the middle ear might lead to grave consequences.

The presence of exostoses is usually easily determined by the cautious use of the probe, with good reflected light, when they are distinguished by their hardness, comparative paleness, and circumscribed form. An exostosis may in some cases be confounded with a furunculus in the ear, but only by inexperienced observers.

**Treatment of Aurial Exostoses.**—In many cases, where the growths do not close the meatus, no treatment need be adopted. The contracted

lumen is, however, apt from time to time to be completely closed by epidermic or ceruminous collection, producing deafness. These should be removed as described at p. 471. The softening and removal of the masses situated beyond the exostosis may be facilitated by syringing through a fine elastic tube introduced through the narrow space. In this way operative treatment may be indefinitely postponed. If a purulent condition exists, either in the middle ear or in the meatus itself, appropriate treatment should be employed. When the meatus becomes entirely blocked by the bony growths, operative treatment is necessary, not only to relieve the deafness and subjective sounds which exist, but also to avert the risk of purulent formation and retention in the deeper parts.

**Methods of Operating.**—A general anæsthetic is necessary. If the exostosis has a slender pedicle it may be able to remove it by encircling the growth, if that be possible, with a Jarvis nasal snare. The author succeeded with a galvanic snare in a case where the pedicle was of considerable thickness. In these pedunculated cases fine but strong dentist's forceps may be used with success, or a tap or two of a mallet upon a suitable chisel may be sufficient to bring the growth away.

Many operators now employ a drill or bur propelled by a dental engine or an electromotor. A variety of burs and drills should be at hand, a speculum may be unnecessary if the exostoses are pretty accessible, but good light reflected into the ear is essential. The tragus is pressed well forward, and if possible a fine steel guard is introduced behind the growth. Owing to the bleeding, frequent mopping with absorbent cotton-wool is required, and the operation may thus occupy a considerable time. If we have to deal with the multiple, hard variety, the apices should be ground away by a small bur till a sufficiently large opening is made. When the growth is large, round, and of a softer texture, its base should first be perforated with a small drill. Till the healing process is complete, narrow strips of iodoform gauze should be pretty firmly packed into the meatus so as to keep the canal open. In many cases, however, it is safer to expose the bony growth, by first making an incision behind the auricle, to the extent of an inch and a half down to the bone, and then displace the auricle and cartilaginous meatus so as to freely expose the bony growth. In this way the bur can be employed more safely than in operation through the meatus. Some prefer, after exposing the exostosis, to use a chisel and mallet behind the base of the growth. The auricle is replaced, and the wound carefully sutured, while the meatus is treated by strips of gauze, as already mentioned. The author has found this a very safe and satisfactory operation.

7 EPITHELIOMA OF THE EXTERNAL MEATUS.—

When occurring in this part of the body epithelioma usually first shows itself by a simple-looking abrasion, going on to thickening of the floor of the cartilaginous meatus. This is followed by ulceration, with surrounding swelling and induration, involving after a time the tragus, the mastoid tissues, and the auricle. There is a constant fetid discharge, while unhealthy-looking granulations sprout up. Pain of an intense and persistent nature is a most prominent symptom, frequently preventing any sleep. Suspicion is aroused as to the true nature of the disease by the fact that the usual remedies have no effect on the pain, discharge, or swelling. As time goes on the whole organ of hearing becomes involved, including the Fallopiian canal, while the cranial cavity is ultimately invaded, and the bones in the neighbourhood of the ear may be extensively exposed. There is facial paralysis, followed eventually by meningeal or cerebral symptoms. Death usually results after from one to two years.

**Treatment**—If the case be seen in the very early stage, thorough excision of all visible disease should be attempted, but, unfortunately, before coming under observation, it has usually extended so far that operative treatment is scarcely admissible. Then regular antiseptic cleansing, with soothing remedies, is all that can be done.

#### Ear: Affections of Tympanic Membrane

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**ACUTE INFLAMMATION**—Acute inflammation of the tympanic membrane, or myringitis acuta, is by no means common as a primary condition. In some cases of otitis externa, and in almost all examples of otitis media, the drumhead participates to some extent, but such are described under "External and Middle Ear." Apart from this, however, it sometimes happens that the membrane becomes inflamed owing to injury caused by the insertion of instruments, or by irritants such as cold or chemicals. Violent syringing is also sometimes responsible for acute myringitis.

The most prominent symptom is severe pain, and this may be associated with tinnitus of a hammering character. Deafness, as we shall presently see, is not very marked when the inflammation is confined to the drum membrane, on the other hand, a certain amount of constitutional disturbance and fever may exist. On examining the ear the membrane is seen to be congested in the early stages, the parts chiefly affected being the immediate neighbourhood of

the malleus and the periphery, while the deeper portion of the meatus may also be involved. At a later stage the whole surface becomes uniformly reddened, and the outline of the malleus is no longer to be distinguished. Sometimes serous and hæmorrhagic blisters, and even small abscesses, appear on the surface, but more frequently, after the stage of uniform redness, the surface epithelium becomes sodden and thrown off in white flakes. Eventually the affection may lead to perforation.

The diagnosis depends upon the symptoms and the objective changes. The only cases which ought to be diagnosed as acute myringitis are those in which the above-described changes in the membrane exist, associated only with slight deafness. There is a method by which diagnosis can be assured, but which should not be employed during the acute stage, as it is then hurtful. I refer to passing the Eustachian catheter while the ear of the surgeon is connected with that of the patient by means of an auscultating tube. In a case of simple myringitis no moist sounds will be heard on the passage of air, while in acute middle ear inflammation these can be detected. As this proceeding increases the pain, and as it is, moreover, of no importance from a therapeutic point of view to differentiate inflammation of the drumhead alone from the same condition associated with acute otitis media, this method should not be practised.

The prognosis is as a rule favourable, even if perforation has occurred. The treatment must be regulated to a great extent by the severity of the symptoms. In any case, it will be desirable to keep the patient in the house, or, if necessary, confine him to bed. Cold applications to the ear may be tried, and, if well tolerated, continued, while leeching over the mastoid region undoubtedly gives relief. As a rule, however, the pain will be readily allayed by the instillation of a few drops of the following solution—

Cocain hydrochlor . . . . . ʒi  
 Acid carbolic . . . . . ʒi  
 Glycerine . . . . . ʒi

or a solution of menthol in paroline up to 20 per cent may be used in the same manner. If these remedies fail to give relief, incision of the inflamed part may be desirable. Of course, if there be evidence of retained serum, blood, or pus, this indication becomes emphasised. So long as the inflammation lasts the meatus should be plugged with sterilised dressing.

**CHRONIC INFLAMMATION OF THE TYMPANIC MEMBRANE**—Various changes in the tympanic membrane, which may be traced to inflammation, are by no means uncommon. Thus in chronic middle ear suppuration a number of different appearances may be met with. Again, in catarrhal and fibroid conditions affecting the tympanum the membrane is frequently implicated. All these are, however, better discussed

in connection with middle ear disease. Prolonged irritation of the external surface may lead to congestion, followed by appearances of dermatitis. Various changes have been described, *e.g.* (1) throwing off of epidermis, (2) the formation of granulations, (3) ulceration, and (4) perforation, the last-mentioned three conditions being associated with more or less suppuration.

According to my own experience the only form of chronic myringitis worthy of practical consideration is that in which the external surface of the drumhead is seen to be thickened, so that the landmarks are more or less obliterated. Even then, however, the malleus can usually be traced, although its outline has lost definition. Sometimes, in addition, scales of epidermis are thrown off, and the appearance is that of a chronic scaly eczema. The treatment must depend upon the result of objective examination. If granulations be present, they should be cauterised with nitrate of silver or chromic acid fused on a probe. The presence of pus indicates careful syringing with boric lotion, followed by drying out and the insufflation of powdered boric acid or instillations of rectified spirit. In the chronic scaly form, which will be often found associated with eczema of the meatus, the application of nitrate of silver dissolved in spirits of nitrous ether (10-20 gr. ad 3j.) will be found effective, while the ear must be kept free from epithelial accumulations by means of the syringe. If such accumulations have already occurred, they may be softened by means of a solution of bicarbonate of soda (15 grs.) in an ounce of equal parts of water and glycerine.

**CHANGES IN THE TYMPANIC MEMBRANE WHICH FOLLOW PREVIOUS INFLAMMATIONS.**—These conditions will be fully described in another part of this work (p. 483). I shall, therefore, merely refer to the fact that a cicatrix always indicates that there has at one time been a perforation, due possibly to myringitis, but probably to middle ear suppuration. Again, the presence of calcareous deposits, although not pathognomonic, is strongly suggestive of previous acute or chronic suppuration. Atrophic patches, on the other hand, indicate chronic middle ear catarrh.

**NEW GROWTHS AND CHRONIC INFECTIVE GRANULOMATA.**—In certain cases aural polypi seem to be attached to the margins of a perforation, but I suspect that this is a very rare occurrence, as these growths commonly have their origins within the tympanum.

Horny growths, cholesteatomata, vascular tumours, osseous deposits, cysts, gummata, and tubercle have been described. The last named is usually associated with a tuberculous condition of the middle ear. In such cases the membrane may show yellow nodules which break down and lead to destruction of tissue, thus increasing the size of the already existing perforation.

**INJURIES OF THE TYMPANIC MEMBRANE.**—The drum membrane may be injured by direct violence. This some persons are fond of boring in their ears with instruments of various kinds, and a sudden accident may drive the extremity against or through the membrane. Such lesions are most commonly found in the anterior segment. A not uncommon form of lesion is found in cases in which abortive attempts have been made with instruments to remove real or imagined foreign bodies by unskilled persons, while rarely one meets with cases in which boiling liquids, having been poured into the ears, have produced burns, followed by perforation. Sudden changes of air pressure either on the inner or outer side may lead to perforation of the membrane. Thus in chalky and cicatrised drumheads the use of Politzer's bag may be followed by perforation, and the same may result from coughing, sneezing, or blowing the nose. Again, boxing the ears, sudden loud sounds, diving, etc., may lead to the same result.

Fractures involving the osseous meatus, and frequently the base of the skull as well, may lead to injury of the tympanic membrane. In some cases of direct violence not only is the drumhead perforated, but the handle of the malleus may be fractured. The symptoms vary considerably according to the cause, and more particularly according to the amount of violence used.

In most instances of the occurrence of a perforation, or even of an injury short of perforation, a sensation of sound is experienced at the moment, associated, of course, with a good deal of pain. This pain may pass off quickly, but, on the other hand, if infection of the tympanum has occurred, it continues, and the case runs the course usual in acute otitis media. There may also be a good deal of bleeding, and the hearing power is always impaired, but to a varying extent. A certain amount of vertigo is by no means uncommon.

On examining the ear after injury to the membrane, the appearances, of course, vary within very wide limits. If only a superficial lesion has resulted, we usually find a localised area of inflammation, frequently associated with the presence of coagula or exudates. If such coagula be watched from day to day it will be seen that they are gradually moved towards the periphery of the membrane, and then on to the wall of the meatus, a phenomenon probably due to eccentric growth of the epithelium. When an actual perforation has taken place, the shape and size of the orifice will vary according to the instrument with which the lesion has been inflicted in cases of direct violence. If the orifice be small it may be covered by clotted blood, while if it be large extravasations and coagula will be detected in the surrounding parts. On the whole, it will probably be best, in cases where doubt exists as to the presence or

absence of a perforation, not to allow diagnostic seal free play Obviously an invisible orifice may be rendered visible by removing blood which covers it, but in doing so the surgeon may infect the middle ear, and produce acute inflammation of the tympanum Again, by Val-salva's method, or by employing the Eustachian catheter, the presence of a perforation can be detected, but under most circumstances it is safer in these cases to trust to sight

The most common forms of perforation, such as are produced by blows, loud sounds, explosions, etc., are usually seen as somewhat lozenge-shaped openings This conformation is produced by the fact that the breach is usually in the circular fibres of the membrane and parallel to the radiating fibres Sometimes instead of being elongated the opening becomes circular In either case the pale yellow mucosa of the tympanum may be seen through the perforation As a result of severe explosions very extensive injuries are sometimes seen Thus I can recall an instance in which both membranes presented irregular rents, so that actual flaps were turned or folded over

The prognosis depends mainly upon two questions, viz (1) whether secondary infection of the tympanum has been avoided, (2) the amount of deafness which exists

If, unfortunately, an acute middle ear inflammation has resulted, the prospects of the patient must be judged by the rules applicable to this disease If the hearing power be only slightly impaired, we may feel fairly confident that it will soon become completely restored On the other hand, should marked deafness be present, and more particularly if bone conduction be impaired, the prognosis should be guarded, at least until obvious improvement has begun to manifest itself In uncomplicated cases the perforation gradually heals, the time occupied varying according to size and shape Where perforation of the membrane is associated with fracture of the base, the importance of the latter, of course, completely overshadows the ear lesion Treatment in traumatic perforation of the membrane is chiefly negative It is of the utmost importance after the injury to avoid syringing, instillations, and the like The ear should as soon as possible be plugged with sterilised wool or gauze The patient must keep quiet for a day or two, and avoid alcohol and tobacco Should pain occur a cold compress may be applied over the ear Should inflammation of the middle ear set in, the treatment recommended in another section must be carried out

**MEDICO-LEGAL ASPECTS**—In examining a case of traumatic perforation from a medico-legal point of view, several points have to be considered Thus, if the injured membrane be cicatrised, and more particularly if there be calcareous deposits, it may safely be assumed that a relatively small amount of violence may

have sufficed to cause a rupture Again, if an injury has been followed by middle ear suppuration, the surgeon will no longer be able to differentiate this from a case due to other causes The history, when accurately obtainable, will be of assistance, but it is well also to examine the uninjured ear If there be evidence of past or present suppuration, this will weigh in favour of the affection on the other side not being traumatic Another important feature is the amount of deafness and the presence or absence of bone-conduction If the hearing be much impaired, and if bone-conduction be lost, the injury must be considered as serious It is then, of course, incumbent upon the medical man to exclude malingering, which may be prompted by a desire for heavy damages It is also well always to make a careful objective and subjective examination

### Ear: Acute Inflammation of the Middle Ear

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**DEFINITION**—Acute inflammation of the middle ear (otitis media acuta) is a more or less severe inflammatory process affecting the whole or some portion of the mucous membrane lining the middle ear, tending either to resolution or to the formation of pus, and sometimes resulting in serious complications and the extension of the inflammation to important neighbouring parts

**INTRODUCTION**—Without entering fully into the anatomy of this region it will be advisable to define very briefly what is meant by the "middle ear," and to draw attention to one or two anatomical points which have an important bearing upon some of the complications which may result from a middle ear attack

The term "middle ear" comprises not only the tympanic cavity, but also the mastoid antrum, the mastoid cells, and the Eustachian canal These parts are in communication with one another, and with the naso-pharynx, by means of the Eustachian tube, and are separated from the external auditory meatus by the tympanic membrane The tympanum, or "drum of the ear," is a narrow, irregular space in the substance of the temporal bone, and is placed between the external meatus and the labyrinth Extending across it is a bridge of small bones, articulated together, which serve to bring the membrane, the outer boundary of the cavity, into communication with the sound-perceiving apparatus These bones are known

as the ossicles—the malleus, the incus, and the stapes. The head of the malleus, and the body and short process of the incus, are situated higher than the upper margin of the membrane, and occupy what is called the upper tympanic cavity or attic. Along with their attachments and some folds of mucous membrane they help to subdivide this cavity into a number of pockets, the lowest of which is known as Prussak's space, which has as its outer boundary the membrana flaccida, or Shrapnell's membrane. Inflammation and suppuration affecting this region is apt to be very intractable and troublesome to treat. On the inner wall of the tympanum will be found the fenestra ovalis or opening into the vestibule, and the fenestra rotunda or opening into the cochlea, both of which are closed by membranes. The roof of the tympanum, or tegmen tympani, is a thin layer of bone forming in part of the middle cranial fossa, which is in apposition with the dura mater on its upper surface. Opposite to the middle and anterior part of the tympanic membrane is a smooth, rounded projection known as the promontory. In front of this we meet with the entrance to the osseous part of the Eustachian tube, while behind and above the fenestra ovalis is part of the bony canal which contains the facial nerve.

An opening in the upper part of the posterior wall of the tympanum communicates with the mastoid antrum, a cavity of varying size. Its roof is continuous with that of the tympanum, and is separated from the dura mater by the same plate of bone, which is sometimes very thin. The floor of the antrum is at a lower level than its opening of communication with the tympanum, and hence secretion does not drain with ease from it. Lower down and more superficially are a number of small pneumatic spaces known as the mastoid cells, and occupying the mastoid process. They vary much in size in different individuals and at different periods of life.

The relations of the middle ear to neighbouring structures, such as the brain, the sigmoid sinus, the facial nerve, and the inner ear, require very careful consideration when we remember the possibility of the extension of inflammation to them, but they will require more detailed notice when the complications of chronic suppurative inflammation and their operative treatment are under consideration.

The somewhat complex cavities forming the middle ear are lined throughout with mucous membrane, which is continuous through the Eustachian canal with that of the naso-pharynx. The diseases of the middle ear will be seen to be due to varying degrees of catarrh or inflammation of this mucous lining, and the disturbances of the function of the organ of hearing associated with them are generally caused by the pathological changes which result from these processes.

Middle ear inflammation is of importance, not only on account of its great frequency, but also because it is liable to affect permanently the function of the organ to a very serious extent, or to produce complications of an exceedingly grave character. According to Gruber, symptoms of otitis media were present in about two-thirds of all ear cases seen in his out-patient clinic. While acute affections probably account for only a small proportion of these, it is evident that the bulk of them must have commenced, at some period or other, with more or less acute symptoms.

**CLASSIFICATION.**—Various attempts have been made to classify acute inflammations of the middle ear on a clinical or pathological basis, but without any very practical result. It is very usual to divide them into acute catarrh and acute suppurative inflammation, and it has also been proposed to separate them into a mild type and a severe type. These divisions do not seem to cover quite the same ground, for an acute catarrh, as often seen in children, may be very severe, while a suppurative attack may be of quite a mild type. Although the onset of a serous or purulent discharge does establish a fact which gives a distinct indication for treatment, yet the classification into perforative and non-perforative cases does not find favour, for it gives no real information as to the course or severity of the individual case.

We propose, therefore, on clinical grounds, to subdivide acute otitis media under the following headings:—

1 Acute catarrh of the middle ear without effusion.

2 Acute inflammation of the middle ear with mucous effusion. In this a perforation may or may not occur.

3 Acute purulent inflammation of the middle ear. In this a perforation always occurs.

As will be seen later on, these are really different stages of the same disease, and any individual case may stop short at the earlier stages, or may pass through them with such rapidity that it is only the latter stage that is recognised.

It is not uncommon to find that authors devote a separate chapter to the consideration of inflammation of the membrane (myringitis). Primary myringitis may be produced by direct exposure or injury of the membrane, or may follow eczema of the meatus, extension of inflammation in the external auditory canal, or may result from foreign bodies or plugs of wax pressing on the membrane, but it is doubtful if the inflammation so set up can be limited for any length of time to the membrane. We shall therefore consider acute inflammation of the membrane as in reality an inflammation of the cavity of the tympanum, possibly differing somewhat in extent and degree.

**CAUSES.**—Acute middle ear catarrh is common in damp, cold climates. It is frequently met

with in early life and in those whose occupation necessitates exposure. Heredity, the gouty, rheumatic, tuberculous, and strumous diatheses, and previous attacks, seem to be predisposing causes. It may result from local conditions affecting the ear primarily, but in far the larger number of cases it spreads by contiguity from local or general conditions affecting the nose and naso-pharynx.

Among the conditions affecting the ear directly we may mention injury to the membrane from a blow on the ear, or from unskilful efforts with instruments to remove plugs of wax and foreign bodies, or from the attempt to relieve irritation in the meatus by means of pins, etc., or from the instillation of very hot lotions, caustics, and corrosive drugs suggested by kind but injudicious friends. The mischief may be stated by a cold wind blowing on the ear during a railway journey or while driving, or by water getting into the meatus during washing or sea-bathing. Reflex irritation due to the eruption of teeth in children, or when the teeth are diseased, has sometimes a large share in producing and keeping up attacks of middle ear catarrh. Among the less frequent causes of otitis may be mentioned fracture through the temporal bone, intra-tympanic hæmorrhage in Bright's disease, and mycosis and fungulosis of the external meatus (*vide* p. 473). By far the larger number of cases, however, are due to morbid conditions of the nose and naso-pharynx. Any of the various conditions which tend to cause and keep up a catarrh of the naso-pharynx, such as an ordinary coryza, hypertrophied tonsils, adenoid vegetations, influenza, diphtheria, mumps, and many others, frequently prove to be the exciting cause of an otitis media. The rhinitis associated with the exanthemata often tends to affect the middle ear, and scarlet fever and measles are specially prone to produce acute purulent otitis. Next to these two diseases adenoid vegetations probably account for the largest number of the less severe attacks.

When patients are described as being specially liable to attacks of middle ear catarrh, it does not follow that there is some hereditary or inherent condition in the ear to account for this tendency, but it will almost invariably be found, on investigation, that some morbid state of the nasal or pharyngeal mucous membrane exists, the removal of which will go far to prevent the recurrence of the attacks.

Various micro-organisms have been discovered in the discharge coming from the ear, and there can be little doubt that they have an important influence in causing and maintaining the diseased condition. The organisms chiefly met with, according to the observations of Löwenberg, Zaufal, Moos, and Netter, are the following:—(1) the streptococcus pyogenes, (2) the staphylococcus pyogenes, (3) the pneumococcus

of Frankel, (4) the pneumo-bacillus of Friedlander, and (5) the tubercle bacillus. Orne Green records the results of a bacteriological examination made of the first drop of pus obtained after paracentesis in 101 cases of acute suppuration of the tympanum. Pure cultures were obtained in 73, showing staphylococcus in 36, streptococcus in 19, pneumococcus in 10, bacillus diphtherie in 2, and bacillus pyocyaneus in 3 cases. There were also 28 cases of mixed infections.

Investigations made by Leuterer tend to show that cases of otitis media with the pneumococcus are generally milder than those associated with the streptococcus. In the former the duration of suppuration was shorter, the febrile reaction less marked, and the injury to bone, if present, was less pronounced. All the same, he found subdural abscess more frequently with the pneumococcus than with the streptococcus. He attempts to explain this by supposing the smaller destruction of bone caused by the former renders external damage more difficult. The attacks of otitis with the streptococcus are apt to be more violent, with greater damage to bone. When sinus thrombosis occurs it is almost always associated with this organism.

According to Green, who has tabulated cultures made from 144 cases of diseased mastoids, we may find any of the common varieties of microbe in mastoiditis, but the staphylococcus is more frequent than the streptococcus. He does not believe that the special organism is of much importance in this disease, and thinks that vastly more depends on the histological and anatomical peculiarities of the bone than on the variety of the microbe. Zaufal has shown that the normal tympanum in rabbits contains germs, and that organisms are abundant in the naso-pharynx. It is therefore more than likely that, under circumstances favourable to the growth and development of microbes, the middle ear is infected through the Eustachian canal, although it is probable that frequently the organism gains access through a perforated membrane. These interesting investigations, however, do not at present give us much assistance in the classification of, or the practical treatment of our cases, but it may be hoped that further knowledge may lead to something of value, at least as to the treatment and prognosis of protracted cases.

In addition to the causes tending to an extension of catarrh through the Eustachian canal, it must be remembered that ear disease may be set up by Politzer's bag or the catheter, used in an improper way or under unsuitable circumstances, and without doubt acute otitis results not infrequently from the careless use of the nasal douche. Fluid may also be forced up the Eustachian tube when diving or swimming, or may pass directly into the middle ear if there be an old perforation.

**PATHOLOGY**—In the early stage of the disease, and throughout the course of a mild case, the catarrh is confined to the superficial layer of the lining membrane of the middle ear. Congestive swelling takes place, and is soon followed by exudation of serum and mucus. In severe forms the inflammation and swelling become more intense, and the deeper perosteal layers are involved, while the exudation becomes purulent in character. The swelling is due to dilated vessels, along with the interstitial, serous, and cellular infiltration. As the case proceeds the epithelial layer tends to soften, and the surface presents a red, soft, granular appearance. Perforation of the membrane is likely to take place in a severe case, either from the direct pressure of the exudation, or from destruction of part of the membrane owing to the acuteness of the inflammation. In the milder attacks the inflammation would appear to be limited in its activity to the Eustachian tube and lower part of the tympanic cavity, but in severe cases the whole lining membrane, including that of the mastoid cells, is equally affected. When this is the case the swelling of the mucous membrane may easily block the normal channels of communication between the various parts of the middle ear, interfering with proper drainage, and thus leading to retention of pus, peristitis, and even necrosis in the mastoid and attic.

**SYMPTOMS**—The prominent symptoms of acute otitis media are pain, deafness, tinnitus, with a varying amount of constitutional disturbance, as shown by a rise of temperature, general malaise, nausea, giddiness, and headache, but these vary greatly according to the type and severity of the attack.

Pain is usually the first symptom to attract attention. It commences as a feeling of discomfort or tension in the ear, which soon becomes actual pain. It is not as a rule continuous, but will almost disappear for a few hours, shortly to reappear with renewed intensity, and this most frequently at night. It may vary from a dull ache to the most intense agony, and to some extent the violence and character of the inflammation may be gauged by the severity of the pain. A numb feeling or actual pain may radiate over the whole side of the head, and it will generally be found that pressure on the tragus, or movement of the auricle, increases the discomfort. In children attacks of "ear-ache" are often spoken of lightly as if they were one of the necessary ills of childhood, but it should be recognised that these are almost invariably due to a middle ear inflammation, and that even in the mildest attack the hearing may suffer permanently. With the appearance of a discharge immediate relief generally comes, at any rate for a time.

Deafness may be slight at the onset of the attack, and may not attract much notice for a

day or two, more especially if one ear alone be affected. Sounds may be correctly recognised, but as if they proceeded from a distance, and the patient often complains that he hears his own voice with almost painful distinctness and force. As the catarrh extends and the exudation and swelling increase, the loss of hearing power becomes more marked, until it may be almost absolute for external sounds. The tuning-fork will be better heard in contact with the mastoid than when its vibrations are conveyed through the air on the affected side. Occasionally when the inflammation is of a very severe type, as in some cases of scarlet or typhoid fever, an early extension to the labyrinth takes place, causing absolute deafness, which is likely to prove more or less permanent.

Noises of some kind are generally heard in the affected ear by the patient, but these seldom cause the annoyance and distress that tinnitus often does in chronic ear affections. The patient's attention is probably centred on the other acute symptoms, and hence he pays less attention at this stage to the tinnitus. However, he often describes a thumping, beating, or pulsating sound, synchronous with the heart's action, as being present, while later on the sound becomes more steady and continuous, like the escape of steam or the rushing of water. Bubbling or crackling sounds are often noticed from the passage of a little air through the fluid exudation in the tympanic cavity.

Constitutional symptoms are sometimes ushered in by a chill or even by a rigor. In mild attacks the temperature does not rise appreciably, but when the attack is very acute the temperature will range from 100° to 104°. There is a general feeling of uneasiness, loss of appetite, and possibly nausea, with headache and dizziness. In people of a nervous temperament, and specially in children, the general disturbance is often very great.

The objective signs of otitis consist mainly in changes in the normal appearance of the membrane, which can only be detected when it is carefully examined with a speculum and reflected light. In the stage of acute catarrh the surface of the membrane is of a dull opaque colour and wanting in lustre. In severe forms its cutaneous layer exhibits marked injection of the small vessels, or its whole surface may assume a bright red or purplish red colour. When serious exudation is poured out into the tympanum the membrane tends to bulge outwards, and it may not be possible to recognise the handle of the malleus. Sometimes when the exudation is copious the membrane appears transparent and not reddened, and it is possible to distinguish through it the presence of a quantity of fluid, a dark line indicating the level to which the fluid has reached. When the exudation becomes purulent the bulging membrane varies in colour from a tinge of red to a

bright red with a yellowish background, or a yellow spot may indicate the point at which perforation is about to take place. When the membrane is oedematous it is sometimes not easy to recognise a small perforation with the eye, but if there be fluid in the meatus, to which a pulsation is communicated, it is safe to assert that the perforation exists. When extensive necrosis of the membrane has occurred the position and extent of the perforation can generally be made out after the meatus has been dried out. The secretion from the tympanum may be mucous, serum, pus, or blood, but more frequently it consists of a combination of these.

The glands behind and below the ear may be swollen and tender, and the soft parts over the mastoid may be oedematous or slightly reddened. Facial paralysis is occasionally though rarely observed in the course of an acute middle ear catarrh from effusion into the Fallopian canal.

The most severe cases of otitis media occur in the course of attacks of scarlatina, measles, and diphtheria, and it is probably due to the organisms present in these cases that the inflammation is of such severity as to cause rapid and extensive destruction of the membrane, and often the early onset of mastoid or other complications.

In otitis associated with tuberculous disease the symptoms seem to attract less notice than usual, because there is generally but little pain. The appearance of a purulent discharge may be the first intimation that the ear requires attention, all the same, these cases very readily become chronic ones.

Influenza is said by some to modify the symptoms of otitis occurring in its course. A hæmorrhagic form of middle ear disease associated with influenza has been described, and a tendency to persistence of mastoid pain after the local conditions have improved is not uncommon. The latter is probably a neuralgic condition due to the well-known depressing power of the disease. Considering the great frequency of so-called influenza, it would appear that the disease does not tend to cause middle ear inflammations to any greater extent than one would expect, in view of the tendency to catarrh of the air passages which accompanies it. An opposite opinion is, however, held by several writers.

**Results.**—The results of acute otitis vary greatly according to the type and severity of the attack. In a simple acute catarrh the majority of cases tend to recover with almost no special treatment. In catarrh with exudation also the result may be—(1) a complete recovery without perceptible changes, (2) recovery with some adhesions and slight defect in hearing, or (3) a perforation with purulent discharge.

The result of acute purulent inflammation may be—

1. Complete cure, with healing of the membrane.

2. Closing of the perforation by cicatricial tissue and adhesion.

3. Continuance of the perforation with or without a chronic discharge.

4. Certain complications which may be dangerous to life.

We have already mentioned that facial paralysis and labyrinthine deafness may occur in the course of an acute otitis, and to these complications may be added mastoid disease, polypi, meningitis, cerebral abscess, thrombosis of the lateral sinus, and septicæmia. As these are much more frequently the accompaniment of chronic suppuration we shall not further refer to them here. No hard and fast rule can be made to decide when an acute otitis should be classed as a chronic case. A consideration of the symptoms will help to decide this question, but, speaking very generally, when a purulent discharge shows no signs of abating after six weeks or two months under treatment, it may be considered that the case is becoming a chronic one.

**DIAGNOSIS.**—The history of the case, taken along with the symptoms and appearances which have been mentioned, makes the diagnosis of a typical case one of no great difficulty. It is a more difficult matter, sometimes, to make an accurate diagnosis when the appearances are masked by some other ear affection, or when we have superadded some of the more serious complications which may supervene in the course of an otitis media. It should be remembered that pain and deafness, the prominent symptoms, are to be met with in various ear conditions, and hence it is not surprising that the true nature of the case is not always evident at the first glance. For example, in furunculosis we find great pain and deafness if the meatus be much blocked by swelling, and possibly a little discharge, in neuralgia from bad teeth we often have acute pain located in the ear, hyperæmia of the membrane, and slight deafness, and plugs of wax, foreign bodies pressing on the drum, eczema, and inflammation of the external meatus may for a time obscure the real condition. Perforation of the drum is sometimes difficult to detect, but the occurrence of this condition can generally be discovered by inspection of the drum, by noting the secretion, and if necessary listening to the sound produced while air is injected into the tympanum.

In certain cases, where the constitutional symptoms are very severe, the ear may at first escape notice and examination, and typhoid fever or meningitis may be suspected.

In infants acute otitis is not uncommon, but the diagnosis is apt to be overlooked because the little patient is unable to indicate the seat of the pain, and the deafness may pass undiscovered. The general symptoms are often very severe,



including high fever, restlessness, vomiting, and even convulsions, before the ear is placed under suspicion. Hartmann has shown that otitis media in infants is often associated with intestinal disturbances, as indicated by dyspepsia and wasting, and that after paracentesis of the membrane the temperature falls, the disturbance in the digestive organs disappears, and an increase of weight follows. In any infant where there are symptoms of restless discomfort, screaming, feverishness, etc., without evident cause for the same, it is well to make a careful examination of the ears for the presence of a possible otitis. The severity of the constitutional symptoms is probably in part due to anatomical peculiarities in the middle ear in the child, such as the relatively greater size and thickness of the membrane and the incomplete development of the bony parts of the middle ear. Measles and scarlet fever produce such a large number of cases of otitis that it is necessary to be on the look-out for early signs of ear trouble, and by early attention to the throat and nose to try and lessen the risk of middle ear affections. It should be remembered that some authorities consider the discharge from the middle ear in a case of scarlatina as highly infectious. It is not clear, however, how long the discharge may be a source of danger to others.

**PROGNOSIS.**—In estimating the prospects of a patient suffering from acute middle ear catarrh we must remember that the disease may affect the function of the organ, or may produce complications which may endanger the patient's health or even his life. It seldom happens that acute otitis leads directly to a fatal result, but the hearing power is often more or less permanently impaired.

In the mild catarrhal types complete recovery is usually to be expected, but in the acute purulent attacks the intensity of the symptoms will generally bear some relation to the course and effects of the disease. Swelling and pain over the mastoid, continuance of the pain, and fever, with copious discharge, make it probable that the case will be a protracted one, while rapid subsidence of the fever and other symptoms after perforation may be viewed in a favourable light. Needless to say, a patient in a robust condition of health is likely to make a quicker and more satisfactory recovery than one who is the subject of tuberculosis, syphilis, or general debility. The position and size of the perforation will sometimes influence the course of the attack, for a very small perforation, or one situated high up, or in Shrapnell's membrane, may cause the retention of discharge instead of allowing it to pass freely away. While necrosis and mastoid abscess seriously protract the illness and call for prompt measures, in acute cases they are much more amenable to successful operative treatment than chronic cases.

Mild attacks of short duration, often spoken of as "earache," are not infrequently viewed as being too trivial to require any special attention. While they may recover completely without any after effects, it should be remembered that not a few cases of auditory defects discovered in later life can be traced back to former apparently trifling and neglected attacks of otitis. Repeated attacks of earache, however slight, are likely sooner or later to cause damage to the ear, and therefore their cause should be investigated without delay.

It is always well to give a guarded prognosis at first as to the hearing power. If there is very marked deafness, and if there is great destruction of the drum and detachment of the ossicles, or if the labyrinth appears to be secondarily affected, there is likely to be more or less permanent interference with the function of the organ. If improvement commences soon after the cessation of the inflammation, and if this is increased by inflation of the tympanum, a favourable result may be predicted with some amount of confidence.

**TREATMENT.**—The remedies to be employed will depend to some extent on the severity of the symptoms and on the stage which the disease has reached before it comes under observation. The objects to be kept in view are to cure the local affection, to diminish the acuteness of the inflammatory process, to prevent complications, and to remove as far as possible the cause of the attack.

Confinement to the house and to a warm room is always advisable, and if his temperature is much above normal the patient should also be kept in bed. The food should be light and non-stimulating, and alcohol and tobacco must be interdicted. The state of the bowels should be inquired into, and it is a good rule to commence the treatment with a small dose of calomel or grey powder, to be followed by a saline purge in the morning. In children, if the skin is hot and dry, a warm bath and a simple diaphoretic mixture may be ordered, while in cases where there is great restlessness or loss of sleep it may be necessary to give bromide or Dover's powder.

In the mild catarrhal stage these remedies may be all that is required, but if the case proves more severe it will next be necessary to adopt some measures for the relief of pain. For this purpose two or three leeches may be applied over the tragus and the mastoid process, or cold may be applied by means of an ice-bag or a Leiter's coil. Most frequently, however, warmth will be found more comforting to the patient than cold, and it should be used in the form of dry heat—flannel, cotton-wool, or the old-fashioned hot salt-bag,—while poultices and moist applications should be avoided. A few drops of chloroform on cotton-wool, or of some aseptic solution containing a local anæsthetic, may be tried with

advantage, but it is necessary that the medication should come into actual contact with the membrane if it is to ease the pain. The anæsthetic action of pure carbolic acid in glycerine, 1 part in 10, has been highly praised, but as a general rule we have employed the following drops with excellent results in relieving the pain and lessening the local inflammation:—

R Cocain hydrochlor	gr	vj
Acid carbolic	gr.	vj.
Glycerin	℥	ij M.

Ten drops of this mixture can be warmed and put into the ear when the pain is troublesome, care being taken to move the auricle backwards and forwards until the drops have reached the membrane. In addition to this, Gribber's medicated gelatine preparations, or solutions containing morphine, atropine, lead and opium, and many other substances, may be used with advantage. Mustard and other counter-irritants applied over the mastoid may allay pain, but they also may mark the onset of mastoiditis, and therefore are better avoided.

When these measures fail to give relief in a short while, it may be necessary to perform paracentesis of the membrane. The indications for this procedure are great pain and bulging of the membrane from excess of secretion. Even when the presence of exudation is doubtful an incision may be recommended, for it greatly eases the pain, and heals very rapidly if no suppurative condition is found. In a commencing purulent inflammation, and specially in scarlatinal cases, it is well to incise early, for not only is the pain thus alleviated, but there is likely to be less destruction of the substance of the membrane than there will be if we wait for spontaneous rupture.

Paracentesis is performed with a slender, spear-shaped knife fitted to a bent handle, and when using this a good light should always be reflected on to the membrane, and every antiseptic precaution attended to. The incision should be of sufficient size to allow the free escape of the retained secretion, and the point usually selected for it is in the posterior inferior quadrant. In children it will generally be necessary to administer a general anæsthetic, but in adults this can usually be dispensed with, for though the pain is very severe it is only momentary. After the opening has been made the meatus should be plugged with sterilised wool, and the ear should not be syringed at all unless there is an undoubted discharge of pus from the incision.

Whenever the acute symptoms have begun to subside it is well to commence inflating the tympanum by one of the recognised methods, whether a perforation exists or not. This has the effect of opening up the Eustachian tube, restoring the normal pressure of the air in the cavity, and dislodging the viscid secretion from

around the ossicles, while if a rupture has taken place it will drive the exudation from the tympanum into the meatus, from which position it can easily be removed. The inflation by Politzer's method should be continued for some weeks, say two or three times a week, in order to prevent the formation of adhesions, or to stretch them if they have already formed. When one ear alone requires inflation, and this requires to be done for a long time, the force of the air douche may over-distend the membrane of the good ear. This can be guarded against by making the patient press firmly with his finger on the tragus of the sound ear in order to counteract the force of the air, or the Eustachian catheter may be substituted for Politzer's bag.

When the third stage, that of purulent discharge, has become established, whether after spontaneous perforation or paracentesis, it is necessary to syringe the ear with some irritating antiseptic lotion. For general use warm boric lotion will answer best, the meatus being thoroughly dried out with absorbent medicated wool after each occasion. It is hardly necessary to particularise the various lotions that have been recommended for this purpose, it is sufficient to say that almost any antiseptic lotion may be used, provided we keep before us the objects to be attained, viz perfect cleanliness and dryness.

The insufflation of a little finely-powdered boracic acid or iodoform often helps to dry up the secretion, but when the perforation is small it should be avoided, as it may cause a mechanical obstruction to the escape of the discharge. Still, when the perforation is of good size and the discharge has begun to lessen in quantity, this method of treatment is very effective in finally drying up the secretion.

Along with the local treatment it is necessary to attend to any nose or throat condition which may be present. Pharyngitis and naso-pharyngeal catarrh should be promptly treated, and it will often do much good to spray the nose with a mild alkaline solution. It must not be forgotten that adenoids, with hypertrophied tonsils, are the cause of many attacks of acute middle ear catarrh, and the operative treatment of these should only be delayed until the acute ear symptoms have passed off. The removal of the adenoids must be looked upon as one of the most important preventive measures against the recurrence of otitic attacks.

Occasionally pain and swelling over the mastoid may, at a very early period, indicate the appearance of a superficial mastoid abscess, and when this is the case it is proper to make a free incision down to the bone, and a little behind the auricle, in order to evacuate the pus. It must be remembered, however, that in such cases, almost without exception, the mastoid cells will be full of pus, and that a minute sinus communicating with the superficial collection of

pus can be found. The external incision, often spoken of as Wilde's incision, often gives great relief to the patient, but it should be looked upon as only the first step in the operation, which should include the opening of the mastoid process also. If this is not done we are very likely to find that a permanent sinus results, and that caries of bone will follow, necessitating a more extensive operation under much less favourable circumstances.

The diagnosis and treatment of the various complications which have been mentioned as occasionally occurring in acute middle ear inflammation do not require consideration here, as they are much more frequently the accompaniment of chronic suppuration, to which the reader is referred.

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### CHRONIC SUPPURATIVE INFLAMMATION OF THE MIDDLE EAR (OTITIS MEDIA PURULENTA CHRONICA).

#### SYN.—*Pyogenic Otitis Media*

CHRONIC suppurative inflammation of the middle ear is in almost all cases the result of a previous acute inflammatory attack. The same ætiological factors which play a prominent part in the production of acute suppurative middle ear catarrh may consequently be ranked as active causes in the production of the chronic type of the disease.

Of the more important affections which fall under the former heading may be cited the various exanthemata, measles, scarlet fever, scarlatinal diphtheria, small-pox, and such general diseases as diphtheria, mumps, pneumonia, influenza (la grippe), pulmonary phthisis, typhoid fever, malum, bronchitis, Bright's disease, cerebro-spinal meningitis, etc.

The extension of catarrhal affections from the nose, the pharynx, and the naso-pharynx plays also a prominent part in the production of acute middle ear suppuration, and hence in many cases in chronic suppurative attacks also.

Many post-nasal growths, especially post-nasal adenoid vegetations, produce such a degree of Eustachian obstruction as to interfere with the normal physiological action of the tube, and are responsible in large numbers of cases not only in causing acute attacks, but also in keeping up such a degree of irritation as to prevent resolution, and so in promoting chronicity.

Causes acting upon the membrana tympani from without, e.g. draughts, the entrance of water (especially sea-water) into the external auditory meatus, or into the middle ear from a too forcible employment of the nasal douche, injuries (blows, concussions, etc.), the extension of a chronic inflammation of the external auditory meatus (Walt), by at times producing acute inflammatory attacks, are also contributory elements in the production of chronic suppurative catarrh.

In tuberculous disease of the middle ear, the process is regarded by most authorities as assuming a chronic type *ab initio*, that is to say, sthenic symptoms are absent, and perforation of the membrana tympani takes place painlessly, a discharge from the cavity of the middle ear being probably the first indication of the presence of the morbid process.

It is doubtful if, tuberculous lesions excepted, the chronic type of the disease is ever observed without there having been at some period an acute, or at least a semi-acute stage present.

In diabetic patients this preliminary acute stage may be of very short duration, the unfavourable effects of diabetes upon the tissues of the middle ear being well recognised.

*Ætiology in Infants*—Suppurative middle

ear disease, both acute and chronic, is more frequently met with in children than in adults, the proportion being 70 to 30. Various factors are responsible for this. In the first place the exanthemata, which are prolific sources of ear trouble, are more commonly met with in children, as are also affections of the lymphoid structures of the upper respiratory tract, *e.g.* enlarged tonsils, naso-pharyngeal adenoids. Dentition, congenital syphilis, and catarrhal affections are also responsible for the production of otitis media in many infants and young children.

Tuberculous disease of the middle ear and adjoining mastoid cells is also comparatively common, especially amongst the children of the poorer classes of our large cities.

Purulent disease is also at times met with in the middle ears of new-born infants, and may be due to an abnormal metamorphosis of the embryonic mucous tissue which exists normally, or to the passage into the middle ear of liquor amni during forced attempts at respiration.

In children acute attacks of middle ear suppuration are at times ushered in by symptoms of great gravity. In fact, until a discharge from the middle ear takes place such cases are frequently diagnosed as inflammation of the membranes of the brain. The practical difficulties encountered in successfully treating acute inflammatory affections of the middle ear in childhood probably go far to explain why so many cases pass into the chronic stage.

**Predisposing Causes.**—Among the most important predisposing causes may be mentioned hereditary tendency, the possession of the so-called strumous, tuberculous, or syphilitic diathesis, the presence of naso-pharyngeal disease, especially post-nasal adenoid vegetations, and the existence of some pre-existing middle ear affection which, having become latent, is prone to be stirred up to renewed activity under the influence of certain unfavourable circumstances.

#### *Segments of the Middle Ear usually involved*

—Chronic purulent disease most usually attacks the mucosa lining the Eustachian tube, and the atrium of the middle ear. In certain cases it may attack the mucous membrane of the recessus epitympanicus (attic), and remain localised in this portion of the middle ear for varying periods. In the later stages of chronic purulent otitis media involvement of the mucosa lining the mastoid antrum and the adjoining mastoid cells may take place.

Certain anatomical peculiarities in the middle ears of young children deserve consideration on account of their practical and clinical importance.

Thus the membrane tympani occupies a much more horizontal position than it does in adults, and is also relatively thicker and larger. Hence in the examination of the middle ear of a child the auricle should be drawn downwards and somewhat forwards, in the adult upwards and backwards.

The Eustachian tube in the young child is shorter, of somewhat larger calibre, and more horizontally situated than in the adult, and hence acts as a somewhat better drain, and is more easily inflated. The mucosa lining the middle ear is frequently in intimate association with the dura by means of a process of fibrous tissue running through the unossified squamopetrosal fissure.

The mastoid or tympanic antrum is, however, of large size, and is more superficially placed than in the adult. The lateral sinus in young children is separated from the mastoid cells by a bridge of bone which is relatively thicker than in the adult.

**BACTERIOLOGY OF CHRONIC SUPPURATIVE INFLAMMATION.**—The rôle played by micro-organisms in the production of chronic suppurative middle ear disease is a most important one. Whether micro-organisms are to be looked upon as the actual factors in the production of middle ear suppuration, or only as incidental to it, is at present a moot point, but that they play a prominent part, not only in the disease itself, but also in the production of its complications, is undoubted. Aerial contamination of the cavity of the middle ear may readily take place either by way of the Eustachian tube or external auditory meatus. Putrefactive bacilli are present in large numbers in fetid discharge from the middle ear, and are absent in non-fetid discharge, and it is a noticeable and important fact that many cases of what are primarily acute catarrhal inflammations of the tympanic mucosa become purulent only after perforation, and hence aerial contamination, has taken place.

The principal organisms found in purulent discharge from the middle ear are—

- (1) *Staphylococcus pyogenes albus et aureus*,
- (2) *streptococcus pyogenes*, (3) *pneumococcus* (Fraenkel), (4) *pneumo-bacillus* (Friedlander), (5) *bacillus tuberculosus*. Further remarks regarding the relative frequency and importance of these micro-organisms will be found on page 484.

Other organisms, *e.g.* *bacillus tenuis*, *bacillus pyocyaneus*, *staphylococcus cereus albus*, etc., have also been discovered in discharge from the middle ear, but do not appear to have the same causal relation as those previously mentioned.

**PATHOLOGY.**—In the initial stages of acute suppurative middle ear catarrh the characteristic features are distension of the tympanic blood-vessels with outpouring of secretion, and subsequent extravasation of leucocytes, partly into the cavity of the middle ear, and partly into the substance of its mucous membrane. In mild cases large quantities of mucus are exuded into the middle ear. In children the exudation may be mainly serous fluid.

As the inflammatory process becomes chronic this small round-celled infiltration results in the

formation of young connective tissue, with consequent interstitial thickening of the tympanic mucous membrane. Accompanying these changes in the deeper layers of the mucosa, the epithelial covering becomes detached, leading either to areas of ulceration, or to the production of œdematous buds of succulent granulation tissue. As the ulcerative process extends, the underlying bone becomes exposed, and ultimately either carious or necrotic. In certain cases small hyperostoses result. Almost every case of chronic suppurative catarrh is accompanied by perforation of the membrana tympani, through which the secretion from the middle ear escapes into the external auditory meatus. The irritation caused by this outflowing secretion leads to a piling up of epithelial cells upon the margins of the perforation, so that in genuinely chronic disease the edges present a pale, callous, and indurated appearance. Through the open perforation the succulent mucosa is to be seen at times so lax and flabby as to protrude into the external auditory meatus.

As a result of this chronic inflammatory process adhesions may take place either between the membrane itself and the mucosa covering the pars promontoria, or between the individual ossicles binding them together, and so interfering seriously with the transmission of sound waves. Or again the inflammatory process may spread to the labyrinth, producing a transient hyperæmia, or in more severe cases an attack of purulent labyrinthitis, ending in complete destruction of hearing (parotitis).

In certain cases a general atrophy of the mucosa ensues, characterised by a thinning of the membrana tympani, and a disappearance of the normal elements of the mucous membrane. Accompanying these changes within the middle ear are changes of an almost similar nature in the mucosa lining the Eustachian tube, e.g. swelling and œdema, enlargement of the mucous glands, shedding of the epithelial lining, etc. As a result considerable stenosis of the tube may ensue.

**SYMPTOMATOLOGY — Subjective Symptoms.** — Pain, which is usually such a prominent feature in acute attacks, is usually absent in chronic cases, or occurs only as the precursor of some serious complication, or as the result of tension due to obstructed outflow of secretion from blockage of the perforation by masses of granulation tissue, polypi, sequestra, stenosis of the meatus, etc. Acute pyogenic attacks grafted upon a chronic basis occur from such causes as exposure to cold, the entrance of water into the ear, etc., and are accompanied by pain of greater or less severity. In chronic cases, when dull, deep-seated pain is complained of in the head, a suspicion of the involvement of the meninges, the cerebral or cerebellar hemispheres, or of the great venous sinuses should be entertained.

In chronic cases when extension to the mastoid process takes place pain becomes a prominent symptom. In debilitated and anæmic subjects pain referable to the branches of the fifth nerve is frequently complained of, and calls for treatment upon general principles.

**Impairment of Hearing.** — The degree of impairment of hearing in chronic cases varies within wide limits. Thus it may be nearly normal in one case, and almost destroyed in another. The size of the perforation appears to bear little if any relation to the existing degree of deafness. Thus cases occur where with almost complete destruction of the membrane a large percentage of hearing power is retained, and others where the hearing power is almost gone, even when a minute perforation exists.

In cases where the perforation is situated in Shrapnell's membrane, the hearing power may be practically unaffected. The presence of a perforated membrane contributes probably only to a small extent to the existing degree of deafness. Other factors which play an almost more important rôle are —

(1) The presence of secretion in the middle ear, preventing transmission of sound waves, and hampering the action of the ossicula auditus.

(2) Adhesions between the membrane and the adjacent walls of the middle ear, between the ossicula themselves, or between the foot-plate of the stapes and the margins of the fenestra ovalis.

(3) The presence of an unduly œdematous or granular mucous membrane lining the cavity tympani.

(4) Loss or partial destruction of one or more ossicles, leading to a want of continuity in the ossicular chain.

(5) Secondary implication of the labyrinth.

**Tinnitus.** — Subjective noises are not frequent in chronic suppurative catarrh with perforation. When they do occur, they are usually due either to adhesion or to indrawing of the ossicular chain, to the pressure of pent-up secretion upon the fenestra, or to an accompanying affection of the labyrinth.

**Vertigo** is also, not as a rule, complained of, and when present is due to the same causes as are responsible for the production of tinnitus.

**Disturbances of taste and smell**, especially of the former, are frequent, and are due to injury to the chorda tympani nerve in its passage through the middle ear, and to the occasional passage of putrid secretion along the Eustachian tube into the pharynx.

**Objective Appearances.** — The most important indication of chronic suppurative middle ear disease is the presence of a discharge. This discharge may be so abundant as to flow from the external auditory meatus, or may exist in such small quantities as merely to form a film over the remains of the membrana tympani. Its consistence, likewise, varies from a thick

creamy pus to a thin ichorous and irritating fluid. At times it is mixed with much mucus, and is consequently tenacious and stringy, at other times it is blood-stained, especially when granulations or polypi are present. Frequently it has a peculiarly offensive odour, when secondary caries or necrosis coexists. At times it has a bluish colour imparted to it by the presence of the bacillus pyocyaneus, at other times a greenish colour, from the presence of the bacillus fluorescens. It may contain numbers of white, glistening epithelial cells, where cholesteatomatous masses are present in the atticus or the mastoid antrum, and in cases of caries of the surrounding bony walls may be mixed with spicules of disintegrating bone.

**Perforation of Membrane.**—Upon examination the membrane will be found to be perforated. Usually only one perforation exists, at times two, and occasionally even three, are to be seen.

The site of the perforation varies much. Most frequently it is situated in the lower segment of the membrane, but may be found in any part. When situated in Shrapnell's membrane it is associated with suppurative disease of the recessus epitympanicus, and when in the posterior part of Shrapnell's membrane, it is said by some authorities to be an indication of suppurative inflammation of the lining membrane of the mastoid antrum.

The size of the perforation varies within wide limits. It may be so small as to be discernible with difficulty, at other times it may practically involve the whole of the membrane. However large the perforation may be, it is rare to find the membrane absolutely destroyed. Usually a small ring around the area of its attachment is left. That portion of the membrane above the level of the short process of the malleus is also usually preserved.

The condition of the edges of the perforation gives in a rough way some idea of the duration of the suppurative process. Thus, in comparatively recent cases the edges have a fairly vascular pinkish appearance, whereas in genuinely chronic cases they present a thick, indurated, and irregular whitish outline. In exceptional cases the membrane may be imperforate, the discharge escaping through fistulous tracts in the postero-superior meatal wall communicating with adjacent mastoid cells or by way of the Eustachian tube.

The diagnosis of the existence of a perforation is usually made by inspection of the membrane under suitable illumination. In certain cases a pulsating spot will be noted, an almost sure sign of the presence of a perforation. By means of a Siegle's pneumatic speculum it is possible to draw secretion from the cavity of the middle ear through the perforation. If imperforate, suction by means of this speculum causes a movement of the whole membrane, whereas if perforated, suction produces no movement what-

ever. Again, by means of the Valsalvan experiment, inflation with Politzer's bag or the Eustachian catheter, secretion can be blown from the middle ear through the perforation, or in cases of a dry condition of the middle ear various sounds will be heard as air blown into the middle emerges through it. Thus, in cases of small perforations the sound is shrill and the note high-pitched.

**PROGNOSIS.**—The question of prognosis has to be considered in reference to danger to life, cessation of discharge, and improvement in hearing.<sup>1</sup> The duration of the disease, the particular part of the middle ear which is implicated, and the underlying cause of the trouble, afford valuable information in estimating these probabilities. With regard to the danger to life, it may be said that so long as suppurative catarrh of the middle ear is present, so long is the patient liable to the occurrence of various septic complications, any one of which may prove fatal. Even in comparatively simple cases severe intracranial complications may suddenly supervene. As a general rule, the longer the disease has lasted the greater is the risk of bone complications, and hence the greater the risk of septic absorption. Disease within the recessus epitympanicus, especially when accompanied by the presence of a small perforation, is undoubtedly more prone to be followed by intracranial complications than when situated within the atrium and when a large perforation is present. When the result of scarlet fever, scarlatinal diphtheria, tuberculosis, or syphilis, the prognosis is not so good as when it is secondary to catarrhal lesions, naso-pharyngeal disease, etc.

The probabilities of a complete cessation of the discharge are much greater if the disease be confined to the atrium than when it implicates the recessus epitympanicus or adjoining mastoid cells. In this latter situation bone lesions are much more frequently met with, and efficient drainage is much more difficult to secure. The continuance of foetid discharge, after prolonged antiseptic treatment, is strong presumptive evidence of the existence of an accompanying bone lesion. When the discharge is tenacious and ropy, chromicity is apt to be favoured.

Improvement in hearing may be anticipated in those cases where, after inflation with Politzer's bag, the range of perception of sound is at once increased, and especially when it is maintained for some hours, or even days.

When, however, free inflation of the middle ear produces no effect upon the hearing power, and when bone-conduction is diminished, the probabilities are that, even if all suppuration be arrested, there will be little or no increase in audition. Where adhesions bind the membrane to the promontory, or the ossicles to one another,

<sup>1</sup> The relation to "Life Insurance" will be considered under that heading.

their division may be followed by improvement, and even in cases where there is an accompanying diminution in bone-conduction, which, under such circumstances, is probably due to temporary increase of intra-labyrinthine tension.

**TREATMENT**—To successfully treat chronic suppurative middle ear disease the guiding principles should be the obtaining of (1) efficient drainage, and (2) surgical cleanliness.

(1) To secure free drainage, small perforations may have to be enlarged, especially when situated in the upper segments of the membrane. Tufts of granulation tissue and polypi demand early removal. Due attention should also be paid to securing a free action of the Eustachian tube by the removal of post-nasal adenoids or other post-nasal growths, and by attention to any morbid condition of the nasal or pharyngeal mucosa likely to keep up a catarrhal state of the surrounding tissues.

To cleanse the cavity of the middle ear the following plan may be adopted with advantage—

- (1) Inflation of the middle ear
- (2) Syringing of the external meatus with a suitable antiseptic lotion or with sterilised water
- (3) Suction by means of Siegle's speculum
- (4) Inflation again
- (5) Syringing or drying with an antiseptic wool

After careful cleansing, one of several methods may be adopted with the idea of restoring the tympanic mucosa to a healthy state. These methods are—

- (1) The employment of fluid remedies in the form of drops or lotions
- (2) The dry method of treatment by means of insufflation of finely pulverised powders
- (3) The aspiration method by means of gauze tampons

Fluid remedies, from the fact that they are easy of application, are the most frequently employed. To ensure their successful action the lotion introduced into the external meatus (after preliminary cleansing) should be allowed to remain in contact with the tissues for from ten to fifteen minutes two or three times daily. Such drugs as carbolic acid, resorcin, sulphocarbonate of copper or of zinc, alum, nitrate of silver, boric acid, acetate of lead, bichloride of mercury, peroxide of hydrogen, etc., may be used with advantage. So long as a perforation is open, it is advisable to keep a film of antiseptic wool or gauze in the meatus, to prevent aerial contamination of the part, to ward off cold, and to assist in drainage.

The insufflation of powders (boric acid, iodoform, euphorben, aristol, etc.) is useful in cases where the perforation is large and where the amount of discharge is small. After careful cleansing and drying of the part, the powder should be insufflated by means of one of the

many insufflators in use for such a purpose, and should be repeated at least once in every twenty-four hours until the discharge has practically ceased.

Where small perforations exist, or where the discharge is copious, this method of treatment is contra-indicated.

The employment of gauze tampons acts admirably in many cases. When a strip of such a gauze as iodoform or double cyanide is carefully packed into the external meatus, and so down upon the perforated membrane, the capillary action of its fine fibrils sucks up discharge from the middle ear and so acts as a continuous drain, besides protecting the parts from aerial contamination—a most important property. Such gauze tampons should be introduced as often as they get soaked. They are most suitable in cases where the amount of discharge is not very copious, and are unsuitable in acute cases and cases where there is an accompanying otitis externa.

#### COMPLICATIONS OF CHRONIC SUPPURATIVE MIDDLE EAR DISEASE

**Granulations**—These are hyperplastic growths resulting from an over-growth of the tissues of the tympanic mucosa, the result of long-continued congestion. They vary in size from minute excrescences to masses which block up the middle ear, protrude through the perforated membrane, and conceal the edges of the perforation in whole or in part, and are frequently associated with an underlying carious condition of some portion of the tympanic parietes. When hanging from the tegmen tympani they must be carefully distinguished from granulation tissue masses attached to the dura mater and projecting into the cavity of the middle ear through carious defects in its bony roof.

Polypi result also from long-continued irritation, the consequence of chronic congestion or suppurative inflammation of the tympanic mucosa. They may be classified as follows—

- (1) Mucous, (2) fibrous, (3) myomatous, (4) angiomatous, (5) malignant

(1) The mucous variety is by far the commonest form of polypus met with. In size such growths vary immensely, being at times quite small and nodular, at other times occupying the whole of the external auditory meatus, concealing the membrane entirely from view, and even projecting externally. Microscopically they consist of numerous small round cells, connective tissue fibres, thin-walled blood-vessels, and glands. Towards its point of origin the polypus is covered by columnar ciliated epithelium, but towards the surface these columnar ciliated cells are replaced by a stratified epithelial layer. Polypi may be attached to the parietes of the middle ear by one or two roots, and usually spring from either the posterior or the internal wall. At times they arise from the edges of a

perforation or from the surface of the membrana tympani itself. Occasionally they arise from the lining membrane of the antrum or mastoid cells, and more rarely still from the walls of the external auditory meatus, in which latter case they result from an otitis externa of the pars ossea.

(2) Fibrous polypi are much less frequently met with, and consist of dense layers of fibrous tissue covered by tessellated epithelial cells. Ramifying through the fibrous strands are small blood-vessels. They originate from the periosteum lining the tympanic cavity or from that of the external auditory meatus.

(3) Myxomatous polypi are distinctly rare and have the structure of gelatinous mucous tissue.

(4) Angiomatous polypi consist of a dense interweaving of blood-vessels supported by a framework of fibrous tissue covered by stratified epithelium. They are consequently very vascular and bleed freely when removed.

(5) Malignant polypi may be either of a carcinomatous or sarcomatous structure. If the former, they may result as the outcome of prolonged suppurative middle ear disease. They are characterised by a marked tendency to sloughing and to spontaneous hemorrhage. They also rapidly invade adjoining bony structures, producing very extensive destruction of tissue.

Sarcomatous polypi may originate within the middle ear, and when appearing externally may present all the appearances of benign growths. Their rapid recurrence after removal and their tendency to spontaneous hemorrhage should excite suspicion as to their real nature. At times they arise from the fibrous sheath of the auditory nerve or from the fibrous stroma of the dura mater, and invade the middle ear in their progress to the surface.

In the treatment of benign aural polypi the primary indication is to treat the inflamed surface from which they originate. When small, soft, and oedematous, then further progress may be checked by the local application of astringent or caustic preparations. Such drugs as perchloride of iron, nitrate of silver, chromic acid, or tichloroacetic acid may be employed with advantage, the drug being either fused to the end of a suitable aural probe or applied in solution upon the end of a cotton-armed probe. To expedite their destruction it is often advantageous to crush them with an aural crush forceps, or rip as much of the growth away as possible with a suitable forceps, and then to apply some caustic drug to the so-called root or base. When sufficiently large, it is advisable to remove as much of the growth as possible by means of a suitable aural snare, the loop being made to grasp the pedicle as near to its base as possible. Hemorrhage, which is at first fairly profuse, is readily con-

trolled by means of warm syringing or by tampons of some antiseptic gauze. Repeated applications of caustic, *e.g.* chromic acid, should be made to the base of the growth until its disappearance has been assured. Astringent lotions, especially when containing rectified spirits, are valuable adjuncts in the subsequent treatment of the case, acting as they do by dehydrating the tissues and causing the coagulation of their albuminous elements.

The treatment of malignant polypi is, unfortunately, far from satisfactory. Occasionally sarcomatous growths originating within the cavity of the middle ear have been successfully eradicated. The tendency, however, of carcinomatous growths to invade the neighbouring bony structures makes operative interference very undesirable.

*Caries and Necrosis*.—As the result of prolonged suppurative inflammation of the mucous membrane lining the tympanic cavity, or as the result of inflammation of the periosteum covering the mastoid process, caries or necrosis of the surrounding bony parietes may ensue. Usually these affections of the bone commence in early life, and are frequently associated with an underlying tuberculous process. Large portions of the temporal bone may become necrotic and may be exfoliated. Thus sequestra containing the whole or part of the cochlea, the annulus tympanicus, or posterior wall of the mastoid process, are by no means infrequently met with in cases of protracted suppurative catarrh.

In the mastoid region fistulous tracts may lead to the interior of the mastoid cells, or may lead from the posterior wall of the antrum into the groove for the sigmoid sinus. The surrounding bone is commonly discoloured and softened. The bony walls of the aqueductus Fallopi are prone to be affected, leading to exposure of the facial nerve and consequent facial paralysis. In cases of attic suppuration the outer wall of the recessus epitympanicus is frequently softened and eroded. The ossicles, more especially the malleus and the incus, are also frequently found to be carious, and in severe cases of middle ear suppuration are prone to become spontaneously exfoliated. In tuberculous cases the whole of the interior of the mastoid process may become broken down, softened, and carious, a mere shell of bone remaining. The inner wall of the middle ear (the pars promontoria) is also frequently implicated, leading to exposure of the contents of the internal ear. The posterior wall of the external auditory meatus may also become affected, in which case its cutaneous covering becomes swollen and infiltrated. Springing from its carious surface are small masses of exuberant granulation tissue.

The indications of caries and necrosis are subjective and objective. Pain may be present,



and if so, is usually deep-seated and of a peculiar boring nature. Complete loss of hearing may result in those cases where the structures of the internal ear have become secondarily affected. Vertiginous symptoms and tinnitus are also frequent under similar circumstances.

The main objective indication is the fact that the carious or necrotic bone may be seen or felt with a probe. The presence also of exuberant granulation tissue masses, of a vascular nature, and showing a marked tendency to recurrence after removal, is of itself strong presumptive evidence of an underlying carious lesion. The periauricular glands also become enlarged at an early period, especially in tuberculous cases.

The accompanying discharge from the middle ear has also, as a rule, a peculiarly offensive odour, and may contain spicules of bone, which may be felt with the fingers. It is also at times blood-stained, even when no granulation tissue is apparent.

#### SUPPURATIVE DISEASE OF THE RECESSUS EPIIMPANICUS (ATTIC)

Suppuration in this region is held by some to be merely an extension of a generalised purulent inflammation of the cavity tympani, by others to be secondary to a primary inflammation of the mucosa lining the mastoid antrum, or to the extension of such morbid processes as *cozena* or *turunculus* of the external auditory meatus (See diagram, p 456.)

Whatever its actual ætiology may be, the fact remains that disease in this situation is peculiarly intractable to ordinary methods of treatment. The numerous folds of mucosa-membrane which exist in the attic tend to prevent the free escape of pus, whilst the frequent shutting off of this region from the general cavity of the middle ear as the result of inflammatory adhesions explains the uselessness of attempting to wash out the part *per tubum*. The fact also that the accompanying perforation of Shrapnell's membrane is situated high up upon the surface of the membrane, and is also frequently very small, serves to explain the difficulties which attend the free escape of pus from the part. The head of the malleus and the body of the incus, being thus constantly bathed in purulent exudation, are prone to undergo carious degeneration. The adjacent bony parietes also frequently become affected, exfoliation of portions of the annulus tympanicus or outer wall of the attic occasionally taking place.

Perforations of the anterior part of the membrane flaccida are most likely to be associated with a morbid condition of the nose, the Eustachian tube, or the tympanic cavity, central perforations with disease of the external auditory meatus, and perforations in the posterior segment of the membrane with disease of the mastoid antrum.

*Symptoms of Attic Disease*—The objective appearances consist in the presence of a perforation (usually small) in either the anterior or posterior segment of Shrapnell's membrane, purulent secretion oozing from the part, and in the frequent presence of caries of the head of the malleus and body of the incus or of the adjacent parietes, at times visible to the eye, but more frequently to be detected by means of a delicate probe. Buds of granulation tissue are also frequently to be seen protruding through the perforation into the external meatus, and are usually found to be springing from carious foci.

*Subjective Symptoms*—Pain is frequently present, especially when retention of secretion takes place from blocking of the perforation by buds of granulation tissue, cholesteatomatous masses, small sequestra, etc.

Tinnitus and vertigo may also be complained of, and result from increased labyrinthine tension due to pressure of inflammatory products upon the foot-plate of the stapes or to a secondary congestion of the labyrinth.

*Treatment*—In order to efficiently cleanse the recessus epitympanicus some form of intatympanic syringe (Hartmann, Pritchard, Milligan) should be used, the end of the syringe being passed well into the attic under illumination. After thorough cleansing various medicaments may be injected into the part by means of specially constructed intatympanic cannule (Blake, Milligan).

Where the accompanying perforation is very small it may be enlarged with advantage. Insufflation of antiseptic powders (Bezold, Gompertz), tamponading (Gruber), and resection of the outer wall of the attic (Poltzer, Schwartz) by means of specially constructed forceps, have been recommended by various authorities.

The ossicula auditus, if carious (and where local applications after a reasonable trial have failed to arrest purulency), should be excised. By the performance of ossiculectomy not only are definite carious foci removed, but improved drainage is effected and better access obtained for subsequent local medication. After thorough cleansing of the meatus and the instillation of a strong solution of cocaine, the patient being under the influence of a general anæsthetic, the membrane, or it remains, is detached by means of a circular incision. The tendon of the tensor tympani, if still intact, is now divided close to its insertion into the bony process of the malleus, after which its superior ligament is also divided by a fine knife. By means of a delicate incus hook (Kretschmann, Poltzer) the incus is brought down, and the incudo-stapedial attachment separated, when with a fine snare or with a strong pair of angular forceps the two ossicles are removed. In cases where the incus has already disappeared as the result of prolonged suppurative inflammation of the tympanic

mucosa, the malleus may be readily excised by means of Delstanche's *extracteur du marteau*.

As a rule the stapes, even in cases of long-continued suppurative disease of the middle ear, retains its vitality. This is to be explained from the fact that it receives nourishment from two sets of blood-vessels—labyrinthine and tympanic—whilst the malleus and incus are dependent upon one set only, viz the tympanic. Its removal has, however, been recommended under certain circumstances by Kessel, and several American confrères, notably Jack of Boston, have recorded surprisingly good results following its excision.

When parietal caries coexists careful curettement by means of delicate spoons or curettes (Lake) may be attempted. The results of excision of the ossicula are in many cases highly satisfactory, vertiginous symptoms being often entirely relieved and purulency arrested. In those cases, however, where purulency continues unabated, the indication is that deep-seated caries coexists, necessitating the performance of a radical mastoid operation. Deep-seated caries may, however, be very difficult, if not impossible, to diagnose with certainty. It may be suspected in those cases of chronic fetid middle ear suppuration which do not yield to ordinary methods of treatment, also where tufts of granulation tissue reappear constantly after removal, and when deep-seated pains are complained of in and around the middle ear.

#### POST-SUPPURATIVE SEQUELÆ

##### *Dry Perforation of the Membrana Tympani*

—When purulency has come to an end, either spontaneously or as the result of efficient treatment, a dry perforation of the membrana tympani frequently remains. In comparatively recent cases such perforations have sharply defined, thin, and somewhat vascular edges, in cases of old standing the edges present a callous and indurated appearance from the presence of epithelial proliferation.

Such open perforations are a constant menace to the integrity of the structures within the middle ear and to the life of the individual. Attempts should therefore be made to induce cicatrization. In recent cases this may be effected by stimulation of the edges by the application of such agencies as nitrate of silver, dilute chromic acid, tincture of iodine, etc. In genuinely chronic cases the application of trichloroacetic acid (Okounoff) will be found most efficacious, acting as it does by removing all dry and indurated epidermis.

Berthold's myringo-plastic method may also be tried.

Multiple incisions across and at right angles to the edges of the perforation, or a circumferential incision, will be found capable at times of starting a healthy reaction and subsequent cicatrization.

In addition to such local applications, packing the meatus with some antiseptic gauze and the maintenance of rest to the organ will be found to assist the process of repair.

When cicatrization of the membrana tympani cannot be secured, and where there is a marked defect in hearing power, the employment of an artificial drum frequently yields excellent results. No data are, however, forthcoming to indicate the cases in which its employment is likely to be attended with success. The membranes most in use are those suggested by Toynbee and Yearsley. Of the two the latter will be found to give the best results in the majority of cases, and its employment, if reasonable care be used, is unattended with risk. Artificial membranes should be used only in those cases where suppuration has actually ceased or where it is present in very small quantities. Yearsley's cotton pads are made by rolling pieces of absorbent wool into small balls or cylinders, which, when moistened with some antiseptic fluid, should be introduced under illumination by means of a delicate pair of forceps and placed against the remains of the membrane, so as to exert slight pressure upon the head of the stapes or head of the malleus. At first they should be worn for a few hours, the period being gradually prolonged until they are borne with impunity. They should, however, be removed at night and replaced the following morning. It is often advisable to intermit their use for a few days, and it is remarkable that in many cases after such a period of rest the hearing power afterwards is considerably better than before. In cases, where they cause marked irritation and a recrudescence of the suppurative process their use should be interdicted. The patient, after having been shown the method of introducing and of placing the artificial drum *in situ*, rapidly acquires an astonishing dexterity in its manipulation.

In a certain number of cases the cicatricial portion of the membrana tympani will be found to be unduly lax, and as a result not only will audition be interfered with, but the constant movement of the cicatrix proves a source of annoyance to the patient. To relieve this, incisions into the cicatrix, or the collodion plan of treatment, by means of which the lax cicatrix is held in position by a film of collodion, may be employed.

*Adhesions*.—As the result of chronic suppuration of the tympanic mucosa, adhesions may form between the remains of the membrana tympani or a cicatrized membrane and the inner wall of the middle ear, between the ossicula themselves or between the ossicula and the adjacent tympanic parietes, or between the foot-plate of the stapes and the margins of the fenestra ovalis. Varying degrees of deafness, tinnitus, and vertigo may consequently result.

In recent cases such adhesions may be broken down or considerably stretched by means of the air douche, Siegle's speculum, or Dolstanche's masseur. Lucæ's probe, by exerting an intermittent pressure upon the ossicular chain and by producing passive movements, is at times useful, although few patients will tolerate its continuous employment. When practicable, division of cicatricial bands or excision of adherent cicatrices by means of suitable instruments may be tried. Mobilisation of the stapes or trephining of the footplate of the stapes has been said to produce good results in certain cases. Excision of the malleus and incus or of either when embedded in scar tissue or when so adherent to one another or to the adjacent walls of the middle ear as to be practically functionless, is undoubtedly a reasonable procedure and worthy of trial.

In all such intratympanic operations strict antiseptics should be observed.

Calcareous deposits upon the membrane are frequently found in post-suppurative affections of the middle ear. Such deposits may occur upon any part of the membrane. A by no means unusual appearance consists in the presence of two semilunar patches of a white or yellowish-white colour with sharply defined edges, one in front of and one behind the manubrium mallei. Occasionally the whole membrana tensa undergoes calcareous degeneration.

*Hyperostosis*.—As a result of long-continued suppurative middle ear disease the lumen of the external auditory meatus may become stenosed from hyperostotic thickening of its bony walls. The accompanying stenosis may be so great as to seriously impede the free escape of pus from the middle ear and consequently to jeopardise the life of the patient.

*Treatment*.—Attempts may be made to dilate the meatus by means of the introduction of laminaria tents or cylinders of wool soaked in alcoholic solutions of boracic acid, acetate of lead, etc. In severe cases, and where evidences of retention are present, the mastoid antrum should be opened and the posterior wall of the external meatus chiselled away.

*Facial paralysis* may result either from a parenchymatous neuritis of the facial nerve (without interstitial changes) secondary to disease within the tympanic cavity, or may be induced by a curious condition of the bony walls of the aqueductus Fallopi. If the paralysis is due to disease within the middle ear the muscles of the corresponding side of the face become paralysed, if, however, due to central disease, the facial muscles upon the side opposite to the existing ear lesion are the ones implicated. Occasionally bilateral facial paralysis is met with. In tuberculous disease of the middle ear facial paralysis is a frequent and an early symptom.

The indications of facial paralysis are a partial

or complete inability to close the eyelids and a general want of expression (best seen during facial movements) upon the affected side of the face. There is also a marked drawing of the mouth to the opposite side and an inability to whistle. Occasionally the uvula is deflected towards the paralysed side. When the stapedius muscle is paralysed there may be an increase of hearing, and subjective noises are at times complained of. Should the paralysis be of a permanent nature atrophy of the facial muscles and even of the facial bones may take place. Ulceration of the cornea is also an occasional result.

*Treatment* consists in dealing with the primary cause as energetically as possible. To keep up the tone of the facial muscles massage or the application of the galvanic current may be employed. Hypodermic injections of strychnia or its internal administration are occasionally useful. Iodide of potassium, especially in cases with an underlying syphilitic basis, may be given. The frequently repeated application of blistering fluid over the mastoid process is occasionally beneficial.

#### DISEASES OF THE MASTOID PROCESS

*Anatomy of Mastoid Process*.—The shape, size, and form of the mastoid process vary greatly in different individuals and at different periods of life. The external conformation of the process affords no clue as to its internal structure, which may be wholly pneumatic, diploetic, or pneumo-diploetic. At birth the mastoid antrum is about as large as a pea, and is lined by mucous membrane continuous with that of the middle ear. The various groups of mastoid cells become developed subsequently around the antral cavity. According to Cheate, the name "mastoid antrum" is a misnomer, the antrum being really a portion of the middle ear. Hence it should be called "tympanic antrum" (see p. 456).

The antrum itself is a bean-shaped cavity. Its roof is formed by the tegmen tympani. Its floor runs downwards and backwards into the mastoid process. Anteriorly it communicates with the attic-tympanic cavity. Its walls are perforated by the minute openings of the surrounding mastoid cells.

*Mastoid Phlegmons* may ensue either as the result of the inflammatory process spreading from the middle ear along the fibrous trabeculae which connect the tympanic mucosa with the periosteum of the external auditory meatus, and so with that covering the mastoid process, or from an extension along connective-tissue strands of the fibrous sheaths of small blood-vessels which run between the perosteum covering the bone and the mucosa lining the mastoid cells. In children the presence of the unossified petro-mastoid fissure probably facilitates the spread of a deep-seated inflammatory process to the

surface. Pathogenic organisms are thus readily conveyed from deep-seated foci of sepsis to the periosteum covering the mastoid process.

In this connection it is important to bear in mind the large size of the mastoid emissary vein in children. Cases occasionally occur in which an abscess outside the skull and over the exit of this vein directly results from extension from the interior. Such cases may present difficulties in diagnosis, and their true nature may only be revealed on operation.

*Symptoms*.—The initial symptom, as a rule, complained of is pain, at first local, and situated just behind the attachment of the auricle, but soon becoming general over the corresponding side of the head. It is always increased by pressure. Redness, swelling, and œdema of the superimposed tissues soon follow, with the result that the ear gets displaced downwards, forwards, and outwards, and, in cases where the œdema is very marked, appears to stand out prominently from the side of the head. This drooping of the auricle is specially noticeable when the patient is examined from behind. At times the œdema becomes so extensive as to extend forwards over the face, producing chemosis of the lower eyelid, or spreads upwards over the surface of the scalp.

Commensurate with the amount of suppuration under the periosteum is the degree of pain and general discomfort of the patient, who exhibits a marked rise of temperature, a rapid pulse, and the ordinary symptoms of febrile reaction.

Such attacks of pericititis may abort without pus formation, but usually an abscess of varying size forms under the periosteum, and as it increases strips it from the bone over considerable areas. The denseness of the superficial tissues retards spontaneous evacuation, which, however, may take place either behind the ear or through the postero-superior wall of the external auditory meatus.

In the early stages of the affection a brisk purgative should be given, the patient being kept within doors and in a warm room. Cold applications, such as ice-cloths, horseshoe-shaped ice caps, or Leiter's continuous cold coil, may be applied behind the ear with advantage.

The local abstraction of blood by means of leeches, or Heurteloup's artificial leech, by diminishing congestion, may bring about resolution. Irritating applications such as acouite, iodine, etc., should be avoided as only tending to mask symptoms. Where pus has formed, and even where it has not, but where the tissues are tense and infiltrated, great relief is afforded by incision. The requisite incision, Wilde's incision, should be made parallel to and  $\frac{1}{2}$  inch behind the attachment of the auricle, and from above downwards for a distance of from  $\frac{3}{4}$  inch to 1 inch. It should be made down to the bone throughout its entire length. It may be neces-

sary to ligate the posterior auricular artery, from which hæmorrhage is usually free, but a fair amount of depletion is to be encouraged as tending to diminish local congestion. Where pointing takes place towards the external auditory meatus its postero-superior wall should be freely incised. The lips of the incision thus made should be kept apart for a few days by means of an antiseptic dressing, after which they may be brought together, provided no other symptoms contra-indicate closure. In cases where mastoid pericititis is secondary to acute inflammation within the mastoid cells, and where symptoms do not subside within forty-eight hours, an opening into the bone should be made. Again, when mastoid pericititis is complicated with a fistula leading to carious bone within the process, suitable treatment must be followed out, *e.g.* enlarging the fistula and scraping away all available disease, or the performance of a radical mastoid operation.

The effect upon the inflammatory process within the middle ear after freely incising the infiltrated tissues over the mastoid process is remarkable. Suppuration, which had previously been abundant, rapidly subsides, with, as a rule, early cessation of the existing perforation.

*Suppurative Endomastoiditis*.—Pathogenic infection of the mucosa lining the mastoid cells exists probably to a greater or less degree in all cases of acute suppurative inflammation of the middle ear. It is only, however, when retention of the inflammatory products within the mastoid antrum or mastoid cells takes place that urgent symptoms arise. Considering the narrowness of the *iter ad antrum*, it is remarkable how seldom such retention does take place in acute cases. When, however, the lining membrane of the walls of the iter become so congested and œdematous as to produce stenosis of the passage, retention of inflammatory products is favoured. Symptoms pointing to such retention are severe pain over the area of the mastoid antrum or infected mastoid cells, increased upon pressure and upon percussion. At times œdema of the superimposed soft tissues takes place, but by no means necessarily. With increasing tension within the mastoid area the temperature rises, and may range from 98° F to 103° F or even more. At the same time other ordinary febrile symptoms are present, *e.g.* rapid pulse, furred tongue, headache, etc.

Upon examination the membrane will in such cases be seen to be swollen, congested, and with its landmarks obliterated. The perforation may be seen to pulsate, and when secretion has been carefully syringed away it will soon be seen to re-collect, indicating that some reservoir exists from which a constant overflow is oozing or flowing away. In addition, the postero-superior wall near its attachment to the membrane will be seen to be prolapsed—the dip,—an almost

pathognomonic sign of involvement of the mastoid cells.

In cases where enlargement of the existing perforation, antiseptic douching, and the local abstraction of blood fail to relieve pain, and where the temperature keeps rising or refuses to fall, opening and drainage of the mastoid cells should be undertaken.

*Preparation of the Patient*—All hair in the immediate neighbourhood of the ear should be shaved, after which the skin should be washed with soap and water and rubbed with ether (so as to get rid of all fatty particles), and then carefully carbolicised. The head should then be wrapped in a carbolic towel, which should be worn for some hours previous to operation.

*Method of Operation (Schwart's Operation)*—When the patient is fully under the influence of an anæsthetic an incision should be made (Wilde's incision) parallel to and a quarter of an inch behind the attachment of the tragus from the linea temporalis to the mastoid apex, and down to the bone throughout its entire length. All bleeding points should be ligated at once, after which, with a raspatory, the periosteum is raised from the bone as far forwards as the posterior border of the bony meatus. The tissues are now held forwards by means of a broad retractor, and the surface of the mastoid process is thus clearly exposed to view.

The object is to open up the cavity of the mastoid antrum and the adjoining mastoid cells, in other words, after having tapped the antrum, to follow up any path of pythogenic infection which may be discovered.

To map out the topographical relations of the mastoid antrum one of two methods may conveniently be employed—

(1) An imaginary line is drawn parallel to and a quarter of an inch behind the posterior border of the bony meatus, and another parallel to its superior border. At the point of intersection of these lines opening of the bone may be commenced, the general direction of the axis of the proposed opening being downwards and forwards parallel to the posterior wall of the external auditory meatus.

(2) *The Supra-Mental Triangle*—Macewen advises opening the antrum in the supra-mental triangle, which is the space formed by the posterior root of the zygomatic process above, the postero-superior margin of the bony meatus in front, and a perpendicular line drawn through the posterior edge of the meatus and joining the lines previously mentioned.

The depth of the antrum from the surface varies greatly, not only in different skulls, but also at different ages and at different stages of the disease. It may be taken, however, as a good practical rule that no exploratory opening should be made to a depth of more than three-quarters of an inch for fear of wounding the external semicircular canal or the facial nerve.

Nowadays either the gouge or the bur driven by a dental engine or an electro-motor is used to lay bare the antrum and adjoining cells. Macewen, the great advocate of the bur, says that by its use safety is ensured, jarring of the intracranial contents is avoided, and a perfectly smooth surface is secured in which the orifices of any fistulous tracts are readily detectable.

With the gouge worked either by the hand or by means of a mallet, layer after layer of bone is removed, a good view of the operation field being thus always obtainable.

Whichever instrument is used good illumination of the field is absolutely requisite, and may be obtained by reflected light from a limelight apparatus or an electric lamp.

It is important to remember that the middle fossa may be found unduly low, that the knee of the lateral sinus may come nearer to the posterior wall of the external auditory meatus than is usual, and that its position may be very superficial, in order to emphasise the necessity of care in all manipulations.

During the removal of the various layers of bone frequent recourse should be made to examination with a pointed probe to ascertain the extent and the size of the various cells which may be opened. When once the antrum has been tapped its extent and depth may readily be gauged by means of the antrum hook. All overhanging bone must now be gouged or buried away, all infected cells opened up, and a cone-shaped cavity formed, with its base superficial.

In cases of acute antral empyema it is neither necessary nor advisable to open up the cavity of the middle ear. The cone-shaped cavity produced as above described should be carefully dried (all syringing being avoided), dusted with some antiseptic powder, and packed loosely with an antiseptic gauze. Healing by means of granulation tissue *ab initio* should be encouraged, and in uncomplicated cases takes place within a few weeks.

*CHRONIC SUPPURATIVE ENDOMASTOIDITIS*—Suppurative disease arising within the middle ear is prone to extend to the mucosa lining the antrum mastoideum and the adjacent mastoid cells. Owing to the complicated arrangement of these cells the free egress of pus is frequently interfered with and chronicity is favoured. Pathogenic organisms also find a suitable habitat within these recesses, and consequently thrive luxuriantly. The mucosa of the part, serving as it does the function of a perosteal lining, tends to undergo ulcerative changes. The underlying bone thus becomes denuded and loses its vitality. Caries, necrosis, or cano necrosis consequently frequently result. In this way large portions of the temporal bone may exfoliate. The most frequent sites for such cario-necrotic processes are the outer wall of the mastoid, the posterior wall of the external osseous meatus, the bony groove for the sigmoid

sinus, the roof of the middle ear, the roof of the mastoid antrum, the pars promontoria, and the bony capsule of the facial nerve. The osseous partitions separating the various mastoid cells from one another become gradually disintegrated, with the result that cavities of varying size form within the mastoid process full of purulent debris, granulation tissue, and cholesteatomatous material. In other cases, however, instead of a rarefying osteitis taking place, a condensing osteitis may ensue, with the result that the individual mastoid cells become obliterated, a mass of dense ivory-like bone being formed.

The subjective symptoms of the presence of caries or necrosis within the mastoid process are comparatively unimportant. Occasionally pain may be complained of, especially when an acute pythogenic infection is grafted upon an already existing chronic process, or where as the result of osseous erosion some intracranial complication is set up. As a result of the presence of a sequestrum, or as the result of the presence of caries of the labyrinthine walls, tinnitus and vertigo may be complained of.

The objective appearances are, however, very important, and consist in the presence of (1) a more or less copious discharge, usually very fetid, occasionally blood-stained, and frequently containing small spicules of disintegrated bone (2) granulation-tissue masses, which when removed are prone to recur, (3) enlarged periauricular lymphatic glands, and (4) the frequent presence of facial paralysis.

A diagnosis of deep-seated caries or necrosis may be made by ocular inspection or by palpation with a suitable probe. Occasionally a positive diagnosis is impossible, but the presumption is in favour of the existence of bone disease when in a chronic case continuous antiseptic cleansing of the part fails to arrest discharge and fetor.

By means of a probe carious foci may be felt. The sensation imparted is that of roughness, combined with a certain degree of softness. Where definite sequestra exist they may be felt to be mobile.

**Treatment**—Thorough cleansing of the diseased areas is imperative, but it is not by any means always attainable without recourse to operative measures. Where the external meatus is stenosed owing to disease of its posterior wall, with infiltration of the soft tissues, specially fine syringes may be required for irrigation purposes, or it may be necessary to dilate the canal by means of cylinders of wool soaked in acetate of lead solution or rubber tubes of gradually increasing calibre. Tufts of exuberant granulation tissue growing from carious foci should be scraped away so as to allow of remedial agents acting upon the underlying disease. Sequestra when sufficiently mobile should be removed by forceps or scoops. The main indications for opening the mastoid process when affected by

chronic inflammatory disease may be summarised as follows.—

(1) In cases where caries of the tympanic walls exists.

(2) When recurring attacks of mastoiditis are present, especially if complicated by the presence of a mastoid fistula.

(3) When a mastoid fistula is present leading down to carious bone.

(4) In cases of cholesteatomata.

(5) In cases of hyperostotic stenosis of the external auditory meatus.

(6) In cases of obstinate mastoid neuralgia (the removal of a wedge of bone is usually sufficient in these cases).

(7) In cases of tuberculosis of the middle ear or mastoid process.

(8) In cases of protracted and futile suppurative inflammation of the middle ear which have resisted the ordinary methods of local treatment.

This indication has led of late to much serious discussion, many eminent authorities claiming that protracted suppuration *per se* is not a sufficient indication for operation in the absence of urgent symptoms. Professor Macceen regards the operation of opening the mastoid as the safest and the most efficient method of eradicating otherwise persistent purulent otitis media.

(9) In all cases of suspected intracranial suppuration the mastoid antrum should be opened as the first step in the operation.

The methods of operation most frequently in use are those designed by Schwartze and Stacke. A combination of the two mentioned known as the Schwartze-Stacke operation has been much in vogue of late years, and has yielded when properly performed very gratifying results.

**Preparation of Patient**—By means of a dose of aperient medicine the bowels are well opened the day prior to the operation.

**Preparation of Operation Field**—The hair for several inches round the affected mastoid should be shaved, the skin well washed with soap and water, rubbed with turpentine, and finally with sulphuric ether. The external auditory meatus should be well syringed with warm carbolic lotion (1-20) or corrosive sublimate solution (1-2000), and packed with iodoform or double cyanide gauze. A pad of lint soaked in carbolic lotion (1-40) and covered by mackintosh should be fixed over the head and kept *in situ* for some hours previous to the operation.

**Preparation of Instruments**—All instruments to be used during the operation should be rendered aseptic by previous boiling, and should be left soaking in weak carbolic lotion or in a solution of formalin for a few hours prior to being used.

The hands of the operator and his assistants should be well washed, rubbed with turpentine, and dipped for a few moments in some warm antiseptic solution.

*Position of Patient*—The patient should lie upon a narrow table of a convenient height, the head reclining upon a firm pillow. Some good source of illumination should be at hand (e.g. limelight, electric light, etc.), so that by means of a forehead mirror the operator may reflect a strong beam of light into the opening in the bone. Some operators use the mallet and the gouge to open the antrum and the adjoining cells; others (Macewou, Ballance) prefer the bur worked by a suitable motor, claiming that by its use there is no risk of concussion, and that a cavity with perfectly smooth walls is left behind. Whichever operation be selected, and by whichever set of instruments the bone be opened, the object to be attained is to open up the antrum and adjoining mastoid cells, to follow the course of any pyogenic lesions which may present themselves, opening up if need be the groove for the lateral sinus, inspecting minutely the roof of the antrum, attic, and middle ear, and finally establishing an efficient and avascular barrier between the middle ear and the adjoining vulnerable parts within the cranium.

*Schwartz's Operation*—An incision is made parallel to and  $\frac{1}{4}$  of an inch behind the attachment of the auricle from the linea temporalis to the apex of the mastoid process. Any bleeding points are to be cauterized and tied. The periosteum is now separated from its attachments along the whole length of the incision, and is drawn forwards until the posterior bony margin of the external meatus is brought fully into view.

The position of the mastoid antrum is now to be defined (see p. 497). By means of a gouge and mallet or by means of a rotatory bur the bone is cautiously removed, frequent examination being made with a fine probe until the cavity of the antrum has been reached. Its depth from the surface may vary from  $\frac{1}{2}$  of an inch to  $\frac{3}{4}$  of an inch. Once the cavity of the antrum has been reached its extent can be readily gauged by exploration with an antrum hook. The surrounding bone should now be freely and rapidly cut away until a conical cavity has been formed with its base uppermost and its main axis parallel to the posterior wall of the auditory meatus. Adjoining mastoid cells, if diseased, should be opened and their contents scraped out by means of spoons or curettes. A stream of some antiseptic fluid should then be driven through the atrial cavity, until it emerges freely from the external auditory meatus.

It may be, and frequently is, necessary to enlarge the passage between the antrum and the middle ear, so as to secure free drainage. This may be done by means of small cutting forceps, spoons, or the rotatory bur. Once a perfectly free communication with the middle ear has been established, a drainage tube should be

placed in the antrum and stitched to the edges of the original incision, which may now be sewn up.

Subsequent treatment consists in thorough irrigation of the antrum and middle ear by means of various antiseptic solutions. When all suppuration has ceased and the parts operated upon have remained dry for from ten days to a fortnight the tube may be withdrawn.

Owing to the formation of tufts of granulation tissue, free drainage is occasionally impeded. When such masses form they should be removed either by means of a sharp spoon or by the application of caustic. Occasionally the tendency to their formation is so great that it is advisable to insert a lead plug into the atrial opening instead of a rubber drainage tube, the weight and the pressure of the spigot materially assisting in checking their formation.

*Stacke's Operation*—The incision through the soft parts is the same as in the Schwartz operation, except that it is carried round the auricle to a point just above the temporo-maxillary articulation. After all bleeding points have been secured the periosteum is raised and drawn forward until the bony margin of the external auditory meatus is in full view. By means of a fine raspatory the cartilaginous meatus is now raised from its attachments, and along with the auricle is drawn forwards on to the cheek. In this way the tympanic structures are fully exposed. The outer wall of the attic is now removed until an antrum hook introduced into the attic and drawn outwards meets with no obstruction. All bony prominences should now be carefully bevelled down, and diseased ossicles, granulation-tissue tufts, and carious foci successively dealt with.

The auricle and cartilaginous meatus are now replaced, a drainage tube inserted into the meatus, and the original skin incision carefully sewn up.

*The Schwartz-Stacke operation*, a combination of the two operations just described, is the one now most usually performed. After the usual skin incision and retraction of the soft parts the antrum is opened as above detailed and the posterior wall of the cartilaginous meatus detached from its bony bed. The bridge of bone which separates the antrum from the external meatus should now be cut away, great care being taken whilst removing its deepest portion to avoid injuring the facial nerve or the external semicircular canal. The antro-tympanic cavity thus formed should be carefully cleansed, diseased ossicles, remnants of the membrana tympani, granulation tissue, and carious foci being successfully dealt with. As in the Stacke operation the subsequent treatment consists in endeavouring to secure a permanently dry epithelial lining in the cavities thus exposed. To attain this end the posterior wall of the cartilaginous meatus is split longitudinally along its centre up

to the concha, where a crucial incision is made. In this way two quadrilateral flaps are formed, which are pressed against the bony walls of the antro-tympanic cavity, the one upwards and the other downwards, and kept *in situ* by packing introduced per meatum. From the edges of these skin flaps epidermisation commences, and spreads until the whole cavity becomes papered with a dry cuticular lining. The original skin incision may be sown up, or Gruber's plan of secondary suture may be adopted, by which is meant the insertion of three or four sutures at the time of operation, and their subsequent tightening some few days afterwards. Every four or five days fresh packing should be introduced, great care being taken to fill the cavity completely. Any tendency to the formation of granulation tissue should be combated by the application of chromic acid or nitrate of silver.

As a rule from three to four months will elapse before complete epidermisation takes place.

**POST-INFLUENZAL SUPPURATIVE MASTOIDITIS** -- Suppurative middle ear disease of influenzal origin is characterised by the intensity of the inflammatory process, by a tendency to spread to the tissues within the mastoid, and by rapid destruction of bone. Mastoid empyemata following attacks of la grippe are most frequently located within the apical cells, the antial cavity and the adjoining cells enjoying some peculiar immunity. In such cases pain is apical, and is much aggravated by pressure upon the inner and anterior aspect of the process.

In the treatment of such subcortical abscesses it is necessary to open the apical cells freely, to scrape away all softened bone and succulent granulation tissue, and to connect the antial cavity with this abscess cavity by means of a gutter-shaped trough. Healing by the formation of healthy granulation tissue rapidly follows.

**BEZOLD'S MASTOIDITIS** -- In certain cases of suppurative endonastoiditis perforation takes place through the inner surface of the mastoid process, close to its tip and into the groove of the digastric muscle. Pus consequently may collect under the deep cervical fascia, giving rise to infiltration of the cellular tissues of the neck, with formation of a hard brawny swelling, or it may pass, guided by processes of the deep cervical fascia, downwards towards the mediastinum.

**Treatment** -- Free ablation of the apical mastoid cells is called for in the first place. A counter opening should also be made in the neck and thorough drainage established. Such cases often run a peculiarly protracted course, owing to the practical difficulties encountered in providing efficient surgical drainage.

**CHOLESTEATOMA** -- This condition occurs in cases of old-standing suppurative middle ear disease, and is generally held to result from an ingrowth into the tympanic cavity through a perforated membrane of epithelial cells from the

dermic layer of the external auditory meatus. Virchow, however, regards these epithelial masses as true neoplasms. Such ingrowing epithelium, mixed with crystals of cholesterol and inspissated pus, collects in concentrically arranged masses within the tympanum or mastoid antrum, leading frequently to dilatation of these cavities and at times to perforation of the surrounding bony parietes. Cholesteatomatous masses swarm with organisms, and when stirred up into activity by some intercurrent inflammatory process may give rise to most urgent symptoms, *e.g.* pain, rise of temperature, etc. As a rule surgical treatment has to be resorted to, although occasionally, if the masses are small and situated fairly superficially, they may be washed out by means of an intiatympanic syringe or dislodged by scoops or curettes. The instillation of rectified spirits may be used with the idea of dehydrating the mass, and so of starving the accompanying micro-organisms. Watery solutions should be avoided. Then marked tendency to recur, and the inherent danger which then presence gives rise to, calls as a rule for surgical treatment. Stucke's radical mastoid operation may be performed with advantage. Rheinhardt has suggested, after clearing out all diseased mastoid cells and epithelial masses, the maintenance of a permanent mastoid fistula, so that in the event of any recurrence taking place efficient local treatment may at once be undertaken. Transplantation of flaps of skin into the cholesteatomatous cavity after it has been freely laid open has also been suggested.

#### TUBERCULOUS DISEASE

Tuberculous disease of the middle ear is held by many authorities to run a chronic course *ab initio*. Its presence is characterised by a somewhat sudden onset, without, however, any sthenic symptoms, by a painless perforation of the membrana tympani, and by an early involvement of the peri-audicular lymph glands, and by the frequent presence of early paralysis of the facial nerve.

Examination of the membrane, which presents a pale, swollen, and oedematous appearance, reveals the presence of one or more perforations with thick, succulent, and avascular edges. The accompanying discharge is usually thin, semipurulent, and frequently very fetid. Buds of flabby granulation tissue protrude through the perforation, and are very frequently associated with deep-seated caries either of the pars promontoria or of some portion of the mastoid process.

In all probability the disease may be primary within the middle ear, infection being conveyed *per tubam*.

In cases of advanced phthisis, the middle ear occasionally becomes involved, painless perforation resulting. Such a complication has a bad prognostic indication.



In such cases careful examination of the discharge should be made for bacilli. Should they, however, not be found in the discharges from the middle ear, any accompanying granulation tissue should be removed and examined. Occasionally they may be discovered in this way. Failing this, portions of diseased tissue from the middle ear may be removed and inoculated into guinea-pigs or rabbits. If such experiments be conducted with due precautions to avoid accidental contamination, and if tuberculous disease be set up in the animal so inoculated, the presumption is that the material employed is tuberculous. Tuberculous disease of the middle ear is frequently associated with similar disease within the mastoid process.

In many such cases where the mastoid has been opened for the purposes of treatment, a pulaceous-looking mass will be found filling up the cells, but this material is practically valueless for experimental purposes, consisting as it does of broken down tissue, unspiculated purulent debris, and epithelial cells. When, however, it has been removed by means of a spoon and the underlying bone exposed, it will be seen where the disease is making progress, and from whence a scraping of bone should be taken.

In my experiments I have inserted a fragment of tissue obtained as above described into a guinea-pig's hind leg, just about the knee-joint, all hair having previously been removed by singeing with a platinum knife. A small pocket is now made with a sterilised needle, and the tissue carefully inserted. In a few weeks' time, should the tissue inoculated be tuberculous, the inguinal glands will be found enlarged, and as time goes on the tuberculous virus will be found to have spread over the animal's body, the glands and viscera being attacked in the following order, according to the results obtained by Professor Del'pue —

*During the second week* after inoculation the lymphatic ganglia upon the same side of the body below the diaphragm and the spleen will be found enlarged.

*During the third week*, the liver, the mediastinal, and the bronchial ganglia.

*During the fourth week*, the lungs, the cervical and the axillary ganglia.

*After the fourth week* some of the lymphatic ganglia of the opposite side of the body below the diaphragm become affected, but this takes place extremely slowly, and the sublingual and popliteal glands escape for a considerable time.

Microscopic sections made from these glands, and stained for bacilli, will frequently be found to reveal their presence.

In this way a definite diagnosis of the actual character of the underlying lesion can be made, and the value of the knowledge thus obtained is naturally immense, both as regards prognosis and treatment.

The course of such tuberculous lesions is only

too often a downward one, despite the most elaborate and painstaking treatment. The practical difficulties encountered in removing tuberculous deposits within bone are immense, and in no region of the body are these difficulties greater than when tubercle attacks the temporal bone, for reasons which must be obvious to all.

The complications which have to be feared are (1) meningitis, (2) tuberculous enteritis, (3) general marasmus.

The treatment of such cases must be considered from two points of view, according as it is non-operative or operative. Cases will be met with, especially in infants, where any operative interference will from the first be seen to be hopeless.

Such are the cases where marked debility and emaciation are present, where a advanced facial paralysis and masses of enlarged glands have been early symptoms, and where the discharge is abundant, fetid, and frequently blood-stained. In such cases palliative measures, antiseptic treatment, and, if possible, residence at the seaside, are indicated, but I am bound to say that in the majority of such patients whose cases I have followed an early death has been the usual history. The prognosis in such cases I believe to be essentially bad.

In other cases, however, where the present condition of the patient is good (and often enough it is so), and where the tuberculous lesion may be regarded as primary and local, much can be done by suitable operative interference. It is almost superfluous to say that the first and the main essential is to provide free drainage. This implies opening and cleansing the mastoid cells, and it is a remarkable fact how often in such cases, without any external and objective sign or indication, the mastoid cortex will be found extensively perforated, and a pulaceous mass immediately exposed to view. Under good illumination a very careful toilet of the part should be effected, and this can generally best be done by means of a sharp spoon. All softened and carious bone must be scraped away, and as smooth a cavity left as possible, even if this necessitates laying bare the dura and walls of the lateral sinus. The cavity thus obtained should be allowed to granulate from the bottom, and care must be taken to stimulate any sluggish area by means of applications of chloride of zinc, nitrate of silver, etc. Frequently more than one scraping is necessary as fresh foci of disease appear. In one particular case which came under my treatment some years ago, and where the cause was proved to have been feeding with milk from a tuberculous cow, five separate operations had to be undertaken before the morbid process was eradicated, which, however, it finally was, and the child has now grown up a healthy and sturdy boy. In very many of the cases the middle ear has been so extensively destroyed that its function as an

organ of sense may be disregarded. Under such circumstances its contents should be freely curetted, and middle ear, antrum, and mastoid cells thrown into one cavity, and allowed to become obliterated by means of healthy granulation tissue. Where, however, a fair degree of hearing is present, efforts should be made to preserve the function of the organ so far as is possible.

An important point arises in connection with the treatment of the accompanying large glands. Some of the glands may be enlarged purely as the result of septic absorption, and if the morbid cause be removed this enlargement will gradually subside, especially if aided by suitable treatment. But many of the glands are of a tuberculous nature, and are prone to undergo caseous degeneration, while at the same time they are a source of possible systemic infection. Hence I hold that after the mastoid area and the cavity of the middle ear have been attended to, and as soon as the condition of the patient admits of it, another operation should be undertaken with the object of removing these enlarged and tuberculous structures.

The facial paralysis which so often accompanies tuberculous disease of the middle ear is unfortunately usually permanent. Something may, however, be done by facial massage and the internal administration of strychnia to assist in maintaining the tonus of the facial muscles.

General treatment, such as the exhibition of cod-liver oil, iodide of iron, syrup of iodine, etc., is useful, as also is change of air and liberal diet. The general conclusions from a study of these cases may be summarised as follows:—

1 That primary tuberculous disease in and around the middle ear is of fairly frequent occurrence, and that it most usually attacks the children of the poor, especially the poor of our larger cities.

2 That a generalised tuberculous infection may arise from a primary focus within or around the middle ear.

3 That the prognosis in such cases is not very favourable, at least 40 to 50 per cent of the cases succumbing even after operative treatment has been undertaken.

4 That in many of the cases operative interference is contra-indicated, owing to the extent of the existing disease and the asthenic condition of the patients.

5 That when operative interference is feasible, the main object should be to scrape away all available foci of disease and to provide efficient drainage.

6 That the best and the most reliable means of establishing the tuberculous nature of the disease is by means of properly conducted inoculation experiments.

#### INTRACRANIAL COMPLICATIONS FOLLOWING CHRONIC SUPPURATIVE OTITIS MEDIA

Extension of a pathogenic infection from the

cavity of the middle ear or mastoid antrum to the interior of the cranium takes place either as the result of carious destruction of the surrounding bony parietes, or as the result of infection spreading along the minute emissary veins or lymphatic channels which connect the mucosa of the middle ear with the meninges and interior of the brain, or directly through various fissures or defects in the osseous framework of the part.

The most usual sites of carious destruction of the bony parietes of the middle ear are—

(1) The tegmen tympani and roof of the mastoid antrum.

(2) The posterior wall of the mastoid antrum.

(3) The inner wall of the middle ear—pars pinnatoria.

(4) The bony walls of the aqueductus Fallopi.

In certain cases extension to the interior of the cranium takes place without any destruction of surrounding bone, the pathogenic infection being conveyed directly from the septic focus within the middle ear by means of venous or lymphatic channels, a septic thrombosis of these vessels taking place, which in turn is followed by abscess formation.

The intracranial lesions most usually met with secondary to chronic suppurative middle ear disease are (1) extradural abscess, (2) pachymeningitis, (3) suppurative pia-arachnitis, (4) temporo-sphenoidal abscess, (5) cerebellar abscess, (6) thrombosis of the lateral sinus, (7) suppurative encephalitis.

**EXTRA-DURAL ABSCESS.**—The most usual sites for extra-dural abscesses are (1) over the tegmen tympani, (2) over the tegmen anti, and (3) in the neighbourhood of the groove for the sigmoid sinus. Their size varies immensely, being sometimes very minute, at other times being so large as to contain several ounces of pus. The underlying bone is frequently discoloured and carious, and may in exceptional cases become perforated, so that pus collects under the pericranium, constituting what is known as "the shirt-button abscess."

The dura mater limiting the abscess cavity is frequently thickened and studded with tufts of exuberant granulation tissue. At times it is perforated, the abscess cavity communicating either with the arachnoid cavity or directly with an intracranial collection of pus.

The symptoms of an extra-dural abscess are pain, at first local, but frequently becoming generalised, rise of temperature, rapid pulse, nausea, and vomiting, and in advanced cases symptoms of cerebral compression. Should the abscess cavity be situated immediately over or in the neighbourhood of the motor area, symptoms of paresis or paralysis of various muscles or groups of muscles may be noted.

Extra-dural abscesses in the neighbourhood of the groove for the sigmoid sinus are frequently associated with thrombosis of the sinus or with cerebellar abscess.

**PACHYMEMINGITIS** is frequently associated with extra-dural abscess and with suppurative pia-arachnitis (lepto-meningitis). In many cases it is the result of an effort of nature to erect a barrier between a suppurating focus within the middle ear and the adjacent brain tissue, so as to prevent the invasion of micro-organisms. In such cases the dura becomes thickened and granular by the deposit of fresh fibrous tissue within its layers, and adhering to the surrounding bone by plastic exudation. Where the underlying bone is carious and perforated, tufts of granulation tissue may protrude into the cavities of the middle ear and mastoid antrum, and may be at first sight mistaken for anal polypi.

**SUPPURATIVE PIA-ARACHNITIS** (lepto-meningitis) is at times very extensive, spreading over the whole base of the brain, and at times over a considerable portion of the cortex. It is frequently a complication of intracranial abscess or of sinus thrombosis. Occasionally cerebral or cerebellar abscesses rupture into the arachnoid cavity, with the result that diffuse and violent arachnitis is rapidly set up.

*Symptoms of Pache- and Lepto-Meningitis.*

The symptom first complained of is pain, which, from being local, rapidly becomes diffuse and intense. The temperature is elevated, and in uncomplicated cases remains so. Where, however, meningitis is associated with abscess it may be subnormal. The pulse is rapid and high. Vomiting is usually present, and may be quite independent of the taking of food. Constipation is also a prominent symptom, and is frequently associated with a markedly retracted abdomen. In basal meningitis pain at the back of the head and neck and retraction of the head are frequently present. Photophobia, strabismus, and optic neuritis, although by no means constant symptoms, are occasionally present. Cerebral symptoms are mainly of the irritative type, consisting of increased excitability, restlessness, convulsions, and general irritability.

Meningeal symptoms are, however, frequently masked by other intracranial lesions, such as abscess or thrombosis, rendering an exact diagnosis practically impossible.

Paracentesis of the spinal theca occasionally affords useful information. Thus, in cases of cerebral abscess the quantity of albumen in the escaping cerebro-spinal fluid is slightly increased, in meningitis it is markedly so, so much so that more than 1 per cent of albumen indicates the presence of meningitis. Absence of polymorphous leucocytes would indicate absence of any inflammatory condition of the lepto-meninges.

**TEMPORO-SPHENOIDAL ABSCESS.**—The majority of temporo-sphenoidal abscesses occur in young subjects and in those under thirty years of age. They occur with an almost equal frequency upon the right and upon the left side, they are more frequently found in males than in females, the proportion being nearly 2 to 1. As a rule

they follow chronic suppurative lesions in and around the middle ear, although they are met with secondary to acute disease. Occasionally an abscess may occur upon the side opposite to the existing ear lesion. They may be encapsulated within the substance of the temporo-sphenoidal lobe, a zone of healthy brain tissue intervening between the abscess cavity and the ear lesion, or they may be connected with the cavity of the middle ear by means of a fistulous tract through a perforated tegmen tympani. The contents of the abscess cavity consist of thick purulent matter, often greenish in colour, and extremely fetid. In recent cases there may be a distinct lining membrane, in chronic cases a thick pyogenic capsule. The surrounding brain substances may be inflamed, softened, and occasionally necrotic.

An abscess may be dominant within the substance of the brain for many years, until an injury or some increase of pathogenic infection due to exposure, cold, etc., may light up the already existing lesion with disastrous results. In a few cases they spontaneously dry up, or become converted into a caseous mass.

*Symptoms.*—One of the earliest and most important of the symptoms of temporo-sphenoidal abscess is pain. This is frequently complained of directly over the site of the abscess cavity, but may and does occur in any part of the head, hence its actual site has no pathognomonic importance. It is usually of a dull, aching character, increased by pressure, and especially by percussion. After a varying duration, drowsiness and stupor are manifested, the patient exhibiting all the symptoms of sluggish cerebration. Vomiting of the cerebral type is an early and important symptom, and may continue for days. The temperature, which in the initial stages of abscess formation is elevated, soon falls as pressure symptoms increase, until it may become one or two degrees below normal. Should the abscess burst into the cavity of the pia-arachnoid or into one of the ventricles, a rapid rise results. The pulse, which during the early stage of the disease may be rapid tends to fall just as the temperature does, until its beats may number only thirty or forty per minute. The respiration wave tends also to become reduced *pari passu* with a fall in temperature and pulse rate.

Paralysis or paralysis of certain muscles or groups of muscles upon the opposite side of the body is also frequently noted, and is due to pressure upon the motor areas, or upon the motor fibres running through the internal capsule. Paralysis of the third nerve is comparatively frequent, and gives rise to such symptoms as ptosis, dilatation of the pupil, loss of accommodation, and a downward and outward movement of the eyeball. Paralysis of the seventh nerve, affecting the facial muscles of the opposite

side, is at times noted, and is due to cortical involvement.

When the abscess cavity is in the upper and posterior part of the temporo-sphenoidal lobe, sensory aphasia is present, when towards its apex and upon the left side motor aphasia may be noted, due to pressure upon Broca's convolution. Optic neuritis may or may not be present.

As intracranial pressure increases, symptoms of coma ensue. If, however, rupture of the abscess takes place into the ventricle or into the arachnoid cavity, symptoms of extreme excitation arise, accompanied by restlessness, convulsive seizures, and rapid elevation of temperature.

**CEREBELLAR ABSCESS**—About one-third of all recorded intracranial abscesses, secondary to chronic suppurative otitis media, occur within the cerebellum. Then most usual situation is towards the anterior extremity of one or other lateral lobe, they are frequently associated with septic thrombosis of the lateral sinus, or with an extra-dural abscess in the posterior cerebral fossa.

The symptoms which an abscess in the cerebellum gives rise to are very much the same as those produced by an abscess in the cerebrum. Pain, which in temporo-sphenoidal abscesses may occur in almost any part of the head, as before described in cerebellar abscesses, is more frequently occipital. Other symptoms, such as pulse rate, temperature, respiration rate, etc., are governed by the same general principles as hold in temporo-sphenoidal abscesses. Giddiness is, however, an indication of some importance, and in certain cerebellar abscesses is very marked, and its character may prove of localising value. Optic neuritis is frequent, and complete blindness may be noted. Constant yawning has also been noted.

Retraction of the head, intolerance of light, and rapid emaciation are important indications.

In cerebellar abscess sudden death may occur from rupture into the fourth ventricle, or from pressure upon, or adhesion around, the respiratory centre.

**THROMBOSIS OF THE LATERAL SINUS** occurs, as a rule, from carious destruction of the bony walls around the sigmoid sinus. In most cases, as a result of perforation of the posterior bony wall of the mastoid antrum, an extra-dural abscess forms, followed by a phlebitis of its walls. This phlebitis produces a certain degree of venous stasis, which in turn is followed by the formation of a thrombus. The thrombus, lying as it does in intimate relation with a septic focus, rapidly becomes septic and disintegrates, minute particles becoming detached and carried by the blood stream to distant organs, e.g. the lungs, the pleura, the kidneys, or the larger joints, there to set up embolic abscesses, or a general septic intoxication may be induced, followed by septicæmia or pyo-septicæmia.

In a certain number of cases thrombosis may

result without any bone lesion existing, the pathogenic infection being conveyed directly from the septic focus within the middle ear or mastoid cells to the lateral sinus, by way of small emissary veins connecting the mucosa of the one with the fibrous sheaths of the other.

A thrombus which has once formed may, under certain circumstances, become organised, its organisation being followed by the formation of a mass of fibrous tissue with obliteration of the sinus.

**Symptoms**—The symptoms which septic thrombosis of the sigmoid sinus produces are mainly those incident to the septic intoxication which results, with, in addition, symptoms referable to the particular organs in which metastatic deposits may have taken place. Rigors are consequently early and important indications, these rigors may occur frequently during the day, the temperature varying from 103° or 104° to normal or subnormal within a few hours. The pulse rate shows corresponding variations. Pain over the mastoid region in the neighbourhood of the sinus is frequently complained of, and is usually increased by pressure and percussion. Edema of the soft tissues over the mastoid may also occur, and is due to blocking of the emissary mastoid vein.

As the phlebitic process advances, pain is complained of along the course of the internal jugular vein, which may become so thick and infiltrated as to feel like a hard cord. The glands in the immediate neighbourhood of the vein also become swollen and tender.

General symptoms, such as vomiting, diarrhoea, profuse perspiration, and exhaustion, are usually present.

When emboli become detached and arrested within the substance of the lungs septic pneumonia results, frequently followed by pulmonary abscess, and even by gangrene of the part. If the pleura becomes affected diffuse septic pleurisy may ensue.

The thrombotic process may also spread to other intracranial sinuses, e.g. the cavernous, the longitudinal, the petrosal, etc. In exceptional cases a general thrombosis of all the venous sinuses within the cranium may result.

As the result of septic absorption, a condition of pyo-septicæmia may be set up, characterised by severe febrile symptoms, frequent rigors, and marked prostration, and by the development of septic abscesses, especially in and around the larger joints.

#### TREATMENT OF THE VARIOUS INTRACRANIAL COMPLICATIONS OF CHRONIC SUPPURATIVE MIDDLE EAR DISEASE

(For the method of preparing the patient for operation, and for other details of operation, see vol. 1 p. 505.)

**Extra-Dural Abscess**—In the treatment of extra-dural abscesses surgical interference should

be resorted to without delay. A disc of bone should be removed over the site of the abscess and free vent given to all pent-up pus. The abscess cavity may or may not be washed out, according to circumstances, but in any case ample provision should be made for free drainage.

Tufts of granulation tissue springing from the inflamed dura should be scraped away with a sharp spoon.

In cases where an extra-dural abscess is suspected, but where its exact situation is a matter of conjecture, discs of bone should be removed over the most usual sites for its formation, viz. over the tegmen tympani and over the groove for the sigmoid sinus.

*Pachy- and Lepto-Meningitis.* Where a diffuse suppurative inflammation of the covering of the brain has taken place little or nothing is to be expected from treatment. Purgation, depletion by means of leeches, wet-bags to the head, and treatment of a supporting nature are all indicated and should be tried.

A bacteriological examination of the purulent contents of the middle ear should be made, and should the predominant organism present be a streptococcus a fair inference is that the meningeal inflammation is of streptococcal origin. Under such circumstances subcutaneous injections of anti-streptococcal serum may be tried.

Where meningitis is, however, local, early operative interference, *ea* removing the bony framework around the infected area, cleansing the parts thoroughly with antiseptic lotions, and providing free drainage, may bring about an arrest of the inflammatory process.

*Temporo-Sphenoidal Abscess.*—Should a diagnosis of temporo-sphenoidal abscess be made in any given case, removal of a disc of bone and exploration of the abscess cavity should be undertaken without delay. After reflection of the soft parts (*see* vol. 1 p. 520) the trephine pin should be made to enter the squamous portion of the temporal bone  $\frac{1}{2}$  inch behind and above the centre of the external auditory meatus (Otterill trephines  $\frac{1}{2}$  inch behind and 1 inch above the centre of the external auditory meatus). After removal of the disc of bone and incision of the dura (which, if an abscess be present, will bulge into the opening) a pus searcher (Horsley), the blade of a fine knife, or a grooved director should be thrust into the substance of the brain in various directions. If pus be present it will well up along the groove of the director. By means of a fine pair of sinus forceps introduced along the director and carefully opened so as to dilate the tract, the contents of the abscess cavity may be evacuated. A drainage tube of rubber or deerskin (chicken bone is now to be introduced and the abscess cavity carefully washed out with warm boracic or carbolic (1-40) lotion. The original skin flap is now to be brought down (an opening being made for the

drainage tube) and the wound sewn up. The usual dressings are then to be applied.

In certain cases a counter-opening through the tegmen antri or tegmen tympani is highly desirable, providing as it does more efficient drainage and a readier means of cleansing the abscess cavity.

*Cerebellar Abscess.*—In operating for cerebellar abscess, after reflection of the soft parts (*see* vol. 1 p. 520), the pin of the trephine should be introduced into the substance of the occipital bone  $\frac{1}{2}$  inch behind the centre of the external meatus (Reid's base line) and  $\frac{1}{2}$  inch below it.

A disc of bone having been removed, the cerebellum should be explored, just as has been previously detailed in connection with temporo-sphenoidal abscesses, and the after-treatment conducted upon the same lines. Once the abscess cavity has been opened and drained, digital exploration is of great value, as occasionally a secondary abscess in close proximity to the first may be present, and its existence is more readily made out by means of the finger than by any other method.

The difficulties of accurately differentiating between a temporo-sphenoidal and a cerebellar abscess are so great that Percy Dean has suggested a trephine opening which will expose both the posterior part of the temporo-sphenoidal lobe and the anterior part of the cerebellar lobe, whilst at the same time affording ready access to the region of the sigmoid sinus. The pin of the trephine is introduced into the bone  $\frac{1}{2}$  inch behind and  $\frac{1}{4}$  inch above the centre of the external meatus and a disc of bone removed. Special care must be taken to avoid wounding the lateral sinus, which lies immediately under the disc of bone which is being removed. Should an abscess cavity not be found in the temporo-sphenoidal lobe, it is easy to explore the anterior part of the cerebellum through the same trephine opening.

*Lateral Sinus Thrombosis.*—After having opened up and cleansed the mastoid antrum and the adjacent mastoid cells the knee of the lateral sinus should be carefully exposed by removing the bone in its immediate neighbourhood. Usually pus in greater or less quantity will be found surrounding the sinus. After cleansing the part a hypodermic needle should be thrust into the centre of the sinus. If no blood be drawn the presumption is that the sinus is thrombosed (the point of the needle will often be found to have a very fetid smell after such a puncture). Should the sinus be found thrombosed the internal jugular vein should be exposed in the neck by an incision along the anterior border of the sterno-mastoid muscle, tied, and divided. The lateral sinus, after having been exposed for about an inch towards its occipital end, is now slit up and its thrombosed and purulent contents freely scraped away until free hemorrhage takes place. It is then to be packed in the direction of the torcular

with strips of iodoform gauze. Its proximal end should also be scraped and a stream of warm carbolic lotion (1-40) syringed along its course until it flows freely through the divided end of the jugular vein in the neck. Antiseptic dressings are then to be applied in the usual way. The packing within the sinus should be allowed to remain *in situ* for five or six days, and then should be carefully removed. If necessary the sinus may be repacked.

**Septic Encephalitis**—As has already been mentioned, the brain tissue in the neighbourhood of the suppurating focus may itself become inflamed, as is evidenced by oedematous swelling, softening, diffuse suppuration, and increase of intracranial tension.

**Treatment** consists in eradicating where possible the primary cause, in treating the secondary focus of suppuration within the cranium, in antiseptically cleansing the suppurating cerebral surface, and in providing free drainage.

#### Middle Ear Chronic Non-Suppurative Disease.

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Under this title are described all cases of deafness due to middle ear disease, in which the membrana tympani is intact.

Although great advances have of late years been made in separating the different forms, we are as yet unable, in many instances, to draw hard and fast lines between them, thus rendering description extremely difficult and often bewildering. The chief difficulty experienced in studying them is, that the opportunity for pathological examination, in their early stages especially, is rarely obtained.

In adopting the following classification it must be understood that one form often appears to exist with another, and that it must be accepted tentatively for the purpose of description only—

- A. Hypertrophic Catarrh
- B. Atrophic Catarrh
- C. Changes due to variations in pressure
- D. Changes due to deficient blood-supply

Of the two chief forms the *hypertrophic* originates in infancy and childhood, both sexes being equally liable, the ultimate results being seen in later life. Adenoids are almost entirely responsible for the condition. The *atrophic* begins insidiously in young adult and early middle life, although it is occasionally met with much earlier. Young women are the chief sufferers.

The symptoms of both forms in their later stages are closely similar, but, in unimixed cases, the history and signs are widely different, in the *hypertrophic*, definite changes are present in the membrane with Eustachian narrowing and local and general signs of past or present nasal obstruction, in the *atrophic*, on the other hand, little or no gross change is observed in the membrane, there is no Eustachian obstruction, and no apparent cause in the nose or nasopharynx. A consideration of this subject indicates the great importance of due attention being paid to the condition of the upper respiratory passages in early life.

**A. HYPERTROPHIC CATARRH**—This is a disease which has its origin principally in early life, having for its chief characteristics, deafness associated with definite changes in the membrana tympani, and some pathological condition in the nose or nasopharynx.

**Causation**—The causes must be any condition which will—

- (1) Predispose to attacks of acute catarrh
- (2) Tend to make acute catarrh become chronic

- (3) Maintain a chronic catarrh

These causes may be local or general, the local condition above all others is chronic hypertrophy of the naso-pharyngeal tonsil (adenoids), a disease chiefly of childhood and early life, although not uncommon in middle life, and occasionally met with at a much later period. Hypertrophied tonsils (although often associated with adenoids), if they are present alone, will help to maintain a chronic catarrh. Other local causes are—secondary syphilis, true or false hypertrophy of the turbinal bodies, suppuration in accessory cavities of the nose, atrophic rhinitis, nasal polypi, irritation due to noxious fumes, tobacco, etc. The general causes are—exposure to wet and cold, anaemia, tubercle, in fact any disease which lowers the vitality of the organism, rendering infection easy, and hindering return to a normal condition.

**Pathology**—In considering the pathology of this disease we will consider shortly what a catarrh is, and what changes are produced by it. But before doing so, the reader may be reminded that the lining membrane of the middle ear consists of three layers—

- (1) Epithelial
- (2) Sub-epithelial, containing lymphatics, nerves, and, comparatively speaking, large blood-vessels, and

(3) A fibrous, which is adherent to the bone

An acute catarrh is an acute inflammation of a mucous membrane due to either injury or infection (*See p 483*) At present we are unable to say definitely what micro-organism will produce catarrh, but, as far as is known, any pathogenic organism has the power

The immediate result of infection is acute swelling and reddening of the membrane due to the engorgement of vessels and the presence of exudation, especially in the sub-epithelial layer. Exudation is also poured out from the surface, being serous, sero-mucoid, or chiefly mucoid. At this point resolution may take place, leaving no trace, the exudation in the sub-epithelial layers being carried off by the lymphatics, and the vessels returning to their normal size. But if, from some local or general cause, resolution does not occur, the engorgement of vessels continues and more or less exudation persists, that which is poured out from the surface being a marked clinical feature in some cases.

The chronic engorgement of vessels leads to local proliferation, especially of the fibrous tissue in the sub-epithelial layer, this fibrous-tissue proliferation undergoes contraction, the exudation ceases, the epithelial layer by stretching becomes atrophied, and the whole lining membrane becomes ultimately converted into a layer of thick fibrous tissue.

There are therefore four stages which run one into another when the acute period is passed —

(1) Chronic engorgement of vessels with exudation

(2) Resulting proliferation, especially of the fibrous tissue

(3) Contraction of the proliferated fibrous tissue

(4) The ultimate stage of cicatricial condition which may be called post-catarrhal

The results of such changes in the middle ear can be easily imagined when it is remembered that the lining membrane, besides clothing the bony walls and inner aspect of the membrane, forms folds and pockets round the ossicles, then joints, ligaments, and muscles.

At first the ossicles and membrana tympani are hampered by the swollen membrane and the exudation. Later the contraction of the proliferated fibrous tissue causes further and permanent fixation.

The membrana tympani is drawn in by the same cause, added by the non-aeration of the cavity through the Eustachian tube allowing external atmospheric pressure to exert its influence. The folds of lining membrane are converted into fibrous bands, binding down the ossicles to neighbouring walls, the malleus to the outer attic wall, and the stapes to its niche. The tip of the handle of the malleus coming in apposition to the promontory, the opposing epithelial layers become rubbed off and allow of adhesion at this point. The ossicular joints be-

come ankylosed, the muscles fixed. The exudation becomes inspissated or confined in pockets of the lining membrane. The Eustachian tube, sharing the same changes, becomes narrowed. So that an originally pink, moist, thin, somewhat movable lining membrane becomes smooth, white, dry, and thick. Further changes of the lining membrane sometimes occur, such as calcification, fatty degeneration, etc. The tensor tympani and stapedius muscles undergo atrophic degeneration.

As the trouble may be limited to the Eustachian tube, or may involve the whole middle ear tract, each will be considered separately with their symptoms, signs, prognosis, diagnosis, and treatment, as far as possible in their different stages.

(a) *Chronic Eustachian Catarrh*—This may be limited to the orifice of the tube, or extend some distance up the cartilaginous portion. If long continued, changes may take place in the whole tract, these will be considered under the changes produced by variations in pressure (*p 516*).

*Symptoms and Signs*—One or both ears may be affected, if both, one is often worse than the other. Deafness is marked, but may vary from time to time, improving sometimes on swallowing or on blowing the nose, but the improvement soon disappears, or if permanent changes have not occurred, the patient, after suffering for some time, may feel a crack in the ears with subsequent complete restoration of hearing.

On inflation with Politzer's bag or the Eustachian catheter immediate and permanent improvement may occur in the limited early stage, or if the disease is of long standing, especially if it has extended some way along the tube, difficulty may be experienced in getting the tube open, and the resulting improvement, though great at the time, sooner or later disappears. On listening with the auscultation tube during inflation the ear can be heard at first in the distance entering with difficulty, before clearly entering the cavity of the tympanum. In the exudation stage distant bubbling may be at first heard. The patient complains of a distinct feeling of oppression of the head on the side affected, and mental dulness may be felt, especially if both tubes are blocked. Tinnitus of a rushing character is heard. The patient's own voice sounds to him louder on the affected side, and if both ears are implicated it seems as if he were talking into a hollow vessel. The auricle and surrounding parts feel numb when lightly touched. On looking at the membrana tympani all the signs of depression may be seen. If permanent changes in the middle ear have not been produced the pink lining membrane may show through, unless any opacity is present. The white, short process of the malleus is prominent, the handle foreshortened and drawn in somewhat backwards. The folds running

with strips of iodoform gauze. Its proximal end should also be scraped and a stream of warm carbolic lotion (1-40) syringed along its course until it flows freely through the divided end of the jugular vein in the neck. Antiseptic dressings are then to be applied in the usual way. The packing within the sinus should be allowed to remain *in situ* for five or six days, and then should be carefully removed. If necessary the sinus may be repacked.

**Septic Encephalitis**—As has already been mentioned, the brain tissue in the neighbourhood of the suppurating focus may itself become inflamed, as is evidenced by oedematous swelling, softening, diffuse suppuration, and increase of intracranial tension.

**Treatment** consists in eradicating where possible the primary cause, in treating the secondary focus of suppuration within the cranium, in antiseptically cleansing the suppurating cerebral surface, and in providing free drainage.

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UNDER this title are described all cases of deafness due to middle ear disease, in which the membrana tympani is intact.

Although great advances have of late years been made in separating the different forms, we are as yet unable, in many instances, to draw hard and fast lines between them, thus rendering description extremely difficult and often bewildering. The chief difficulty experienced in studying them is, that the opportunity for pathological examination, in their early stages especially, is rarely obtained.

In adopting the following classification it must be understood that one form often appears to exist with another, and that it must be accepted tentatively for the purpose of description only—

- A Hypertrophic Catarrh
- B Atrophic Catarrh
- C Changes due to variations in pressure
- D. Changes due to deficient blood-supply

Of the two chief forms the *hypertrophic* originates in infancy and childhood, both sexes being equally liable, the ultimate results being seen in later life. Adenoids are almost entirely responsible for the condition. The *atrophic* begins insidiously in young adult and early middle life, although it is occasionally met with much earlier. Young women are the chief sufferers.

The symptoms of both forms in their later stages are closely similar, but, in unimixed cases, the history and signs are widely different, in the *hypertrophic*, definite changes are present in the membrane with Eustachian narrowing and local and general signs of past or present nasal obstruction, in the *atrophic*, on the other hand, little or no gross change is observed in the membrane, there is no Eustachian obstruction, and no apparent cause in the nose or nasopharynx. A consideration of this subject indicates the great importance of due attention being paid to the condition of the upper respiratory passages in early life.

**A HYPERTROPHIC CATARRH**—This is a disease which has its origin principally in early life, having for its chief characteristics, deafness associated with definite changes in the membrana tympani, and some pathological condition in the nose or nasopharynx.

**Causation**—The causes must be any condition which will—

- (1) Predispose to attacks of acute catarrh
- (2) Tend to make acute catarrh become chronic

- (3) Maintain a chronic catarrh

These causes may be local or general, the local condition above all others is chronic hypertrophy of the naso-pharyngeal tonsil (adenoids), a disease chiefly of childhood and early life, although not uncommon in middle life, and occasionally met with at a much later period. Hypertrophied tonsils (although often associated with adenoids), if they are present alone, will help to maintain a chronic catarrh. Other local causes are—secondary syphilis, true or false hypertrophy of the tubal bodies, suppuration in accessory cavities of the nose, atrophic rhinitis, nasal polypi, irritation due to noxious fumes, tobacco, etc. The general causes are—exposure to wet and cold, anæmia, tubercle, in fact any disease which lowers the vitality of the organism, rendering infection easy, and hindering return to a normal condition.

**Pathology**—In considering the pathology of this disease we will consider shortly what a catarrh is, and what changes are produced by it. But before doing so, the reader may be reminded that the lining membrane of the middle ear consists of three layers—

- (1) Epithelial
- (2) Sub-epithelial, containing lymphatics, nerves, and, comparatively speaking, large blood-vessels, and



(3) A fibrous, which is adherent to the bone

An acute catarrh is an acute inflammation of a mucous membrane due to either injury or infection (See p 483) At present we are unable to say definitely what micro-organism will produce catarrh, but, as far as is known, any pathogenic organism has the power

The immediate result of infection is acute swelling and reddening of the membrane due to the engorgement of vessels and the presence of exudation, especially in the sub-epithelial layer. Exudation is also poured out from the surface, being serous, sero-mucoid, or chiefly mucoid. At this point resolution may take place, leaving no trace, the exudation in the sub-epithelial layers being carried off by the lymphatics, and the vessels returning to their normal size. But if, from some local or general cause, resolution does not occur, the engorgement of vessels continues and more or less exudation persists, that which is poured out from the surface being a marked clinical feature in some cases.

The chronic engorgement of vessels leads to local proliferation, especially of the fibrous tissue in the sub-epithelial layer. This fibrous-tissue proliferation undergoes contraction, the exudation ceases, the epithelial layer by stretching becomes atrophied, and the whole lining membrane becomes ultimately converted into a layer of thick fibrous tissue.

There are therefore four stages which run one into another when the acute period is passed —

(1) Chronic engorgement of vessels with exudation

(2) Resulting proliferation, especially of the fibrous tissue

(3) Contraction of the proliferated fibrous tissue

(4) The ultimate stage of cicatricial condition which may be called post-catarrhal

The results of such changes in the middle ear can be easily imagined when it is remembered that the lining membrane, besides clothing the bony walls and inner aspect of the membrane, forms folds and pockets round the ossicles, then joints, ligaments, and muscles.

At first the ossicles and membrana tympani are hampered by the swollen membrane and the exudation. Later the contraction of the proliferated fibrous tissue causes further and permanent fixation.

The membrana tympani is drawn in by the same cause, aided by the non-aeration of the cavity through the Eustachian tube allowing external atmospheric pressure to exert its influence. The folds of lining membrane are converted into fibrous bands, binding down the ossicles to neighbouring walls, the malleus to the outer attic wall, and the stapes to its niche. The tip of the handle of the malleus coming in apposition to the promontory, the opposing epithelial layers become rubbed off and allow of adhesion at this point. The ossicular joints be-

come ankylosed, the muscles fixed. The exudation becomes inspissated or confined in pockets of the lining membrane. The Eustachian tube, sharing the same changes, becomes narrowed. So that an originally pink, moist, thin, somewhat movable lining membrane becomes smooth, white, dry, and thick. Further changes of the lining membrane sometimes occur, such as calcification, fatty degeneration, etc. The tensor tympani and stapedius muscles undergo atrophic degeneration.

As the trouble may be limited to the Eustachian tube, or may involve the whole middle ear tract, each will be considered separately, with their symptoms, signs, prognosis, diagnosis, and treatment, as far as possible in their different stages.

(a) *Chronic Eustachian Catarrh*.—This may be limited to the orifice of the tube, or extend some distance up the cartilaginous portion. If long continued, changes may take place in the whole tract, these will be considered under the changes produced by variations in pressure (p 516).

*Symptoms and Signs*.—One or both ears may be affected, if both, one is often worse than the other. Deafness is marked, but may vary from time to time, improving sometimes on swallowing or on blowing the nose, but the improvement soon disappears, or if permanent changes have not occurred, the patient, after suffering for some time, may feel a crack in the ears with subsequent complete restoration of hearing.

On inflation with Politzer's bag or the Eustachian catheter immediate and permanent improvement may occur in the limited early stage, or if the disease is of long standing, especially if it has extended some way along the tube, difficulty may be experienced in getting the tube open, and the resulting improvement, though great at the time, sooner or later disappears. On listening with the auscultation tube during inflation the air can be heard at first in the distance entering with difficulty, before clearly entering the cavity of the tympanum. In the exudation stage distant bubbling may be at first heard. The patient complains of a distinct feeling of oppression of the head on the side affected, and mental dullness may be felt, especially if both tubes are blocked. Tinnitus of a rushing character is heard. The patient's own voice sounds to him louder on the affected side, and if both ears are implicated it seems as if he were talking into a hollow vessel. The auricle and surrounding parts feel numb when lightly touched. On looking at the membrana tympani all the signs of depression will be seen. If permanent changes in the middle ear have not been produced the pink lining membrane may show through, unless any opacity is present. The white, short process of the malleus is prominent, the handle foreshortened and drawn somewhat backwards. The folds running

forwards and backwards from the short process to the periphery are marked, the latter especially. The triangular light reflection from the tip of the handle of the malleus is interrupted, shortened, or absent, depending upon the amount of depression. The structures in the middle ear and the inner wall may be clearly visible. In the posterior and superior segment, the descending process of the incus with the stapedius muscle running backwards from close to its tip, the chorda tympani nerve running from behind upwards and forwards across the descending process of the incus, may be seen. The smooth curve of the promontory in the inferior segment is well marked, and below and behind it the round window appears as a dark patch. On looking at the naso-pharynx a catarrhal condition of the lining membrane may be seen, it being red and swollen with exudation lying on its surface. Sometimes this can be seen involving the lips and orifice of the Eustachian tube, and rarely a plug of exudation may be seen lying in the orifice.

*Prognosis*.—In the early stages this is very good, in the later it will depend on the amount of constriction produced, and whether the tympanum is also involved.

*Diagnosis*.—Simple chronic Eustachian catarrh will be diagnosed by the marked improvement of hearing by inflation and the absence of permanent changes in the membrane. The general methods of diagnosing middle from internal ear affections are described on page 459.

*Treatment*.—Any causes in the nose or naso-pharynx must be removed, and treatment directed to improving the patient's general condition adopted, such as change of air and tonics. In the early stages a single inflation may be all that is necessary to open a tube which has been perhaps blocked by a plug of mucus, or the sides of which have stuck together. If the trouble has extended some distance up the tube, inflation will have to be repeated, the intervals between the proceeding depending on the effect produced and the length of time improvement in hearing remains.

In the later stages, when contraction appears to become a definite feature, especially if the cartilaginous portion is affected, injections of alkaline solution or of paroline, or the passage of a bougie through the Eustachian catheter, may be necessary. Combined with this treatment the chloride of ammonium vapour, obtained by means of Godfrey's or Baston's inhaler, drawn into the mouth and blown through the nose for ten minutes night and morning with a few auto-inflations into the middle ear by means of Valsalva's method when the mouth and nose are full of vapour, is often of great use.

An alkaline and astringent solution gently syringed down the nose after the inhaler is

often useful. In the last stage it will usually be found that further changes in the upper middle ear tract have occurred either by extension of the catarrh, or by changes produced by the variation in pressure. The treatment in these cases will be considered later.

A useful point, when difficulty of opening the Eustachian tube by ordinary inflation is experienced, is to place a few drops of pure chloroform into the bag before inflation.

(b) *Chronic Hypertrophic Catarrh in the whole Middle Ear Tract*.—*Symptoms and Signs*.—These will depend on how far the disease has progressed.

1 *Stage of Chronic Engorgement of Vessels with Exudation*.—Although a certain amount of exudation from the surface is present in all cases, yet in some it forms a very prominent clinical feature demanding special description. It must be stated that cases of this variety are undoubtedly rare in Great Britain. It is impossible to say what determines this excess of exudation, undoubtedly in some cases the excess is more apparent than real, being due to the collection of exudation in the tympanum owing to coexisting Eustachian obstruction. The character of the exudation varies, being serous, sero-mucoid, or mucoid, and here also it is impossible to say definitely on what the varying characters of the exudation depend.

The history of these cases in which *exudation is marked* usually is that, after an acute catarrh of the naso-pharynx, deafness in one or both ears persists. The exudation form may be present on the one side, with simple Eustachian obstruction on the other. There is a feeling of fullness in the ear, stopping short of actual pain, and a sensation of something moving, especially if the exudation is serous, with occasional bubbling, especially after blowing the nose. Improvement in hearing occurs temporarily, but is only of short duration, and may vary with the position of the head. The patient may hear imbibles bursting, especially after inflation. Rushing and occasionally pulsating tinnitus is present. The head on the affected side feels heavy, and numbness of the auricle and surrounding parts is experienced. The patient's own voice sounds unusually loud. Inability to do mental work is often complained of, and sleep may be disturbed, owing to the bubbling and cracking which goes on in the ear. When the mucoid element predominates bubbling and variations on posture are not marked.

Sometimes, especially in old people, the membrane may rupture on blowing the nose or on inflation, producing perhaps a suppurative process resulting from septic infection from the meatus. In infancy and early childhood rupture seems readily to take place in the early stages.

On examination the appearance of the mem-

brane varies according to the character and amount of the exudation, the length of time it has been present, and the degree of clearness of the membrane.

If the exudation is slight and serous the malleol vessels are somewhat injected, and the fluid can be seen occupying the lower part, its upper level, which appears as a thin dark line, varying with the position of the head, or if greater in amount, marked bulging, usually in the posterior segment, is seen, perhaps completely hiding the handle of the malleus. On inflation, a disturbance of the fluid can be seen with the formation of bubbles, or if the auscultation tube be used, clear bubbling can be heard. If the mucoid element preponderates, a whitish-yellow appearance of a more or less bulging membrane is seen with dilated vessels coursing over, there is little or no movement observable on posture, and on inflation through the catheter, as the bag is often not effective, the air can be heard at first in the distance and then gradually to enter the tympanum with sticky rales.

In the later period of the exudative stage the exudation partly escapes from the Eustachian tube, while some becomes inspissated,—the membrane in the meanwhile becoming pale and depressed, with perhaps localised collections of exudation.

In those cases in which *exudation* is not a marked clinical feature the symptoms and signs are not so aggressive. Deafness, improving on blowing the nose, with gradual return to the former condition, and tinnitus of a rushing and pulsating character, are complained of. The membrane is somewhat depressed, the lining membrane seen through being dark pink in colour. On inflation slight bubbling may be heard, the improvement in hearing produced being greater and more lasting than in the cases of marked exudation.

**Prognosis.**—This, if the case be properly taken in hand, is as a rule excellent, but if long-continued or untreated, resulting in further changes in the lining membrane, as demonstrated chiefly by the amount of improvement in hearing produced by efficient inflation, renders it proportionately worse, therefore before giving a definite opinion in long-standing cases it is well to wait the effect of treatment.

**Diagnosis.**—The cases of marked exudation give definite signs.

From a collection of pus they are diagnosed by the absence of acute redness of the membrane, chronicity, and the absence of pain and fever. If the exudation is mucoid, a general whitish appearance is seen instead of a yellow or greenish yellow, which is seen when the contents of the tympanum are purulent. The effect of treatment will also help. In those cases in which exudation is not a marked clinical feature bubbling is sometimes heard,

and the unusually dark and swollen lining membrane seen through the membrana tympani, together with the persistency of the symptoms and effects of inflation, mark them from simple Eustachian obstruction.

**Treatment.**—At this stage, as we have seen, it is possible for the disease to be completely arrested, and no means should be spared to prevent further progress. The treatment is local and general. Those cases in which *exudation* is marked will be first considered.

If this is slight and serous the treatment adopted for simple Eustachian catarrh will suffice. As before stated, it is impossible at first to say how much Eustachian obstruction is responsible for the collection of exudation in the tympanum, sometimes cases which at first appear to demand more radical measures yield to simple treatment. In the cases in which simple remedies do not effect a cure, a collection of exudation persisting, and especially when the mucoid element predominates, they must be supplemented by intra-tympanic injections of warm sterile alkaline solutions, such as bicarbonate of soda, five grains to the ounce, or of paroline. Counter-irritation behind the ear, or massage from above downwards behind the ear and upper part of the neck, may also be used. If these measures do not suffice, the membrane must be opened, perhaps more than once, as the incision speedily closes in spite of inflation. The incision must be made under strict antiseptic precautions, through the part in which the bulging is most marked, or, if no bulging is present, in the posterior and inferior segment, it should be free and parallel to the handle of the malleus. At the time of incision inflation should be practised to clear the middle ear, the exudation being then gently mopped out. The meatus should then be lightly plugged with the antiseptic dressing. The simple treatment of the nose and naso-pharynx with the chloride of ammonium inhaler and nasal solution should be continued meanwhile. Massage by means of Siegle's speculum will be found useful in hastening absorption and preventing adhesions. Change of air to a high and dry climate with tonics is often very beneficial.

In those cases in which *exudation* is not marked, simple treatment, regular inflation, massage of the membrane, together with tonics and change of air, will usually be found sufficient. In these cases, again, local trouble in the nose or naso-pharynx must also be removed.

**2 Stages of Proliferation and Contraction.**—In discussing the following stages they merge so gradually, one into the other, that it is impossible to separate them completely, the progress of the cases being judged according to the amount of improvement obtained by treatment and the changes present in the membrane. We may discuss the stages of proliferation and

contraction together. This is a common period for patients to present themselves for treatment, as they find that the deafness, which they thought would pass off in time, has not only persisted, but is gradually getting worse.

**Symptoms and Signs.**—The history of these patients, usually young adults, is that deafness has persisted after a cold or series of colds, or has gradually come on since, or that in childhood occasional deafness was noticed, with a history that points strongly to the fact that adenoids were present at that period. In fact they often present the appearance due to chronic nasal obstruction. They also state that they are worse with every cold, with perhaps marked permanent deterioration. In the later stage, when far advanced, the symptom of hearing better in a noise may begin to show itself, indicating the gradual onset of the fixation stage. Deafness is well marked, both ears as a rule being affected, one, often the left, being the worse. The fact that the patient cannot hear general conversation, or when at a dinner-party, he cannot hear conversation distinctly on one side, may be the symptom which compels him to come for treatment. Tinnitus, rushing, roaring, clanging, or machinery-like in character, is often a source of great trouble, being worse when the patient is quiet, especially at night, sometimes preventing sleep.

Diploacusis, usually disharmonic, is sometimes complained of. On examination the membrane is pale, often opaque, with perhaps patches of chalky deposit (phosphate of lime), the signs of depression being marked, and in the later stage the pink lining membrane cannot be seen, even if the drum is clear.

On applying Siegle's speculum it will be found that the membrane and malleus do not move freely, or perhaps the posterior segment will alone be freely movable. On inflation through the catheter the air will be heard to enter with difficulty and dryly, with perhaps a whistling sound. The amount of improvement in hearing produced will vary according to how far pathological changes have progressed. On examining the membrane after inflation little or no alteration is seen. The nose or naso-pharynx may present some pathological condition, and it is often possible to detect, even in middle life, remains of adenoids, which if seen during a cold may be considerable in size.

**Prognosis.**—For this we rely upon the progressive character of the deafness, which is worse with each cold, and the absence of internal ear trouble as shown by the tuning-fork, etc. From the exudation stage, by the absence of moist sounds on inflation, the depression, fixation, and opacity of the membrane, and the amount of improvement produced by inflation. From the last stage, by the amount of improvement produced by inflation, the absence of paracusis, which, though present in the later

stages of contraction, appears to indicate that the final stage is being reached.

**Treatment.**—If on inflation the improvement in hearing is marked, we gather that the contraction stage has not advanced far, and we must adopt treatment which will, as much as possible, cut short the proliferation or limit the amount of contraction.

In order to do this, definite local troubles in the naso-pharynx or nose must be removed, followed by regular inflation by means of the bag or Eustachian catheter, the intervals between the inflations being judged by the length of time improvement in hearing lasts. Massage by means of Siegle's speculum or Delstanche's masson is also useful. The chloride of ammonium inhaler and the nasal solution, with tones and change of air to a high and dry climate, should be combined with the other treatment.

If the Eustachian obstruction is a prominent feature a bougie may be passed up the tube, or parolene may be injected through the catheter. If the results produced by this treatment are not great we must infer that contraction is well advanced, and we may have to consider, if the deafness is extreme, the question of operative treatment, which will be presently dealt with in considering the treatment of the post-catarhal stage.

Sometimes iodide of potassium in small doses, combined with ammonia given in hot water twice a day for a fortnight or three weeks, produces good results. With regard to tinnitus, the treatment we have indicated will usually do as much good as is possible, as it is mechanically produced and depends on the local changes. Tones are useful in rendering the patient more able to stand the noises, bromide of potassium and diluted hydrobromic acid are sometimes useful as sedatives. Electricity may be tried if at the first sitting neither the anode nor cathode alters the sound the case is unfavourable, but if the noises are diminished during the passage of the anodal current, the treatment is more hopeful and should be continued. (Lewis Jones, *Archives of Otolaryngology*, vol. xxiv.)

**3. Centraireal or Post-Catarhal Stage.**—This may be looked upon as the ultimate condition resulting from the unchecked progress of the disease, which may have occupied a longer or shorter length of time.

**Symptoms and Signs.**—A history of gradual increasing deafness of catarrhal origin, distinctly worse on colds, until a pitch of deafness has arrived which, although never absolute, necessitates a loud voice close to the ear. Paracusis Willisii, or hearing better in a noise, is a prominent symptom, this phenomenon is supposed to be due to vibration produced by jolting or by loud noises, enabling the rigid ossicular chain to more readily transfer sound waves. Occasionally the patients will state that they

used to hear better in a noise. Tinnitus is often very distressing, sometimes rendering life almost unbearable, and in a few even suicidal tendencies may be present. The patients are morose, introspective, being to a large extent cut off from the outside world. Many acquire in some degree the power of lip reading, and they will consequently hear better when the speaker is facing them. A low but clear voice is heard better than shouting. High tones will be distinctly heard better than low, for instance, a watch will be heard comparatively better than the human voice. On looking at the membrane it will appear markedly depressed and opaque. By means of Siegle's speculum the malleus may be firmly adherent to the promontory, the membrane perhaps fixed to the descending process of the incus and round the malleus to the promontory. On inflation the eardrum with difficulty and produces no change in the position of the malleus or membrane. Improvement in hearing is either absent, or, if slight, is of short duration. Diminution of the noise is sometimes produced.

**Prognosis**—Is extremely bad as regards hearing and tinnitus, deafness is never absolute, and the patient may become more or less used to the noises, which may vary with the state of health. Operative measures may produce improvement, if not in hearing, in tinnitus.

**Diagnosis**—From the previous stages it may be diagnosed by the fixation of the membrane, the obstruction of the tube, the paracusis, and the slight improvement on inflation. The tuning-fork and tone-hearing tests will distinguish it from internal ear disease, but we occasionally find that the tuning-fork in these cases indicates a certain amount of internal ear implication, the history of paracusis will be sufficient to stamp the case as having originated in the middle ear, especially if other symptoms of internal ear disease are absent. True auditory vertigo does not occur.

**Treatment**—It follows from the pathological condition that ordinary local and general treatment is useless in effecting useful or any permanent improvement in hearing or tinnitus. The treatment, however, described under the previous stage should be given a fair trial. If the patient is satisfied with the temporary and slight improvement which may be effected, especially if lip-reading lessons are taken, ordinary methods of treatment should be from time to time employed.

(c) **Operative Measures**—Before undertaking operative measures care must be taken that the internal ear is intact, and it must be pointed out to the patient that they are more or less of an experimental nature. It is well to fully explain to the patient the true condition of things, and to leave it to him to decide as to whether they should be undertaken. It is wise to first operate on the ear which is more affected. These intratympanic operations fall under three headings—

- (1) Those undertaken to relieve tension
- (2) Those undertaken to diminish undue flaccidity

- (3) Those undertaken to allow sound waves to reach the fenestra direct

None of them should be undertaken without strict antiseptic precautions.

(1) *Those undertaken to relieve Tension*—These comprise division of adhesions, section through the posterior fold, tenotomy of the tensor tympani, and division of ligaments. These have not realised expectations, any improvement which is produced speedily disappearing as soon as the inevitable healing takes place, therefore they may be placed on one side.

(2) *Those undertaken in cases of undue flaccidity*—When the membrane or some part of the membrane is seen by inflation or the Siegle's speculum to be unduly flaccid, due to atrophy or to energetic inflations, or the result of a cicatrix, especially if marked improvement in hearing occurs when it is put on the stretch, multiple incisions made through the flaccid part with the idea of producing cicatricial contraction may be undertaken, but the result is often disappointing. Collodion painted over the flaccid portion and adjacent meatal wall may be of benefit.

(3) *Those undertaken to allow of Sound Waves reaching the Fenestra direct*—It has long been known that the artificial perforation of the membrane will in some cases produce great improvement in hearing, but as healing always takes place, and no method of keeping the perforation open has yet been devised, some further procedure becomes necessary. As to whether further proceedings should be adopted exploratory tympanotomy is a useful guide, for if it produces improvement in hearing or tinnitus, we are encouraged to proceed to more radical measures, but even if it does not, and given that the internal ear is intact, and the case is not one of atrophy, we may, if the patient so desires, adopt the more radical measures, as it may mean that the absence of improvement is due to fixation of the stapes, or to blocking of the round window to cicatricial tissue.

**Exploratory Tympanotomy**—This little operation is best performed under gas anaesthesia, by cutting a flap with its apex uppermost in the posterior and superior segment, by means of a sharp-pointed knife, under a good reflected light.

Preliminary inflation of the middle ear may be useful in separating the membrane as far as possible from the middle ear wall. Further procedures can be divided into two stages—

(a) Removal of the incus, malleus, and incus, which if not productive of improvement, even after an artificial membrane has been tried, may be followed by

(b) Mobilisation or removal of the stapes,

and removal of cicatricial tissue from over the round window

(a) *The Removal of the Membrane, Malleus, and Incus.*—This should be performed under a general anæsthetic, the head being slightly raised on a pillow and turned three-quarters over to the opposite side. A good reflected light is necessary. An incision is made with a sharp-pointed knife, starting from immediately behind the short process of the malleus, sweeping round as close to the periphery as possible to a corresponding point on the anterior aspect of the short process. The handle is then freed from adhesions which may be present between the membrane or malleus and the promontory. The tensor tympani is then divided, either by Delstanché's extract or by a small curved knife. The malleus is then seized as high up as possible with a pair of strong curved forceps, being pulled first downwards to free it from the attic, and then outwards. The incus must then be turned out from the attic by means of an incus hook, which, being introduced into the anterior part of the cavity, is rotated downwards and backwards, pushing the ossicle into the lower middle ear, when it may be removed by forceps or by syringing.

Numerous incus hooks are made, the most useful being either Delstanché's, Lake's, or Ludewig's. The middle ear should then be gently mopped out, a gauze dressing should be lightly introduced into the meatus, and a general dressing and bandage applied. If antiseptic precautions have been efficient, dressing will not be required for a week or ten days. At the end of a fortnight or three weeks the hearing power should be tested again, and the amount of tinnitus noticed. The dressing should not be left out until healing is complete, when an artificial membrane may be tried if no improvement results. Sometimes an adventitious membrane forms across, annulling any good effect, and may require removal more than once.

(b) *Mobilisation and the Removal of the Stapes and the Removal of Cicatricial Tissue from over the round Window.*—Before these operations are performed the ear should be allowed to heal soundly, allowing the condition of the inner middle wall to be plainly seen. Adhesions binding down the head and crura of the stapes should be divided with a fine sharp, shouldered knife, such as Politzer's, as close to the ossicle as possible, under eucaine or cocaine, the stapedius muscle being also divided, and the stapes mobilised by means of a suitable probe. If improvement occurs, nothing more should be done, if it does not, we may again try an artificial membrane, if this is ineffective we should remove adhesions obscuring the round window as far as possible, a rather difficult procedure on account of the anatomy of the part. If this is insufficient we must infer fixation of the base of the stapes.

With regard to removal of the stapes more experience and investigation is necessary.

If mobilisation has not been possible, attempted removal will, in all probability, result in fracture of the crura, leaving the foot-plate still in position. The attempted removal should be made by means of a fine hook introduced between the crura from above, and with a gentle side to side movement. It may be that, in the future, operations on the inner middle ear wall may be of benefit. A more radical method has been proposed and carried out by Malherbe, who opens the antrum from behind, divides adhesions in the middle ear, and introduces a celluloid tube through the meatus into the antrum (*Proceedings of the Sixth International Congress of Otolaryngology*, 1899), the results have not been brilliant, and until further experience has been obtained it may be fairly stated that operations through the meatus, as described, are equally efficient.

*B. ATROPHIC CATARRH.—Fixation of Stapes.*—The chief characteristics of this form are the very gradual and insidious onset of deafness, with little or no change in the membrane and no obvious cause in the nose or naso-pharynx, the majority of cases occurring in women between the ages of twenty and forty.

*Causation.*—The causes are obscure in the highest degree. Heredity is certainly an important factor. Occasionally a vague history of a bad cold or series of colds is obtained as a starting-point. Some severe illness, such as rheumatic fever, is thought sometimes by the patient to be the origin, and occasionally chronic rheumatic affections are coexistent. Anæmia is often present. Parturition is intimately connected with this class, the deafness either apparently commencing after labour, or being made considerably and permanently worse thereby.

*Pathology.*—This appears to be an atrophy of the lining membrane with a marked tendency to the fixation of the base of the stapes in the oval window, and sometimes implication of the internal ear in the later stages. The onset is so gradual that pathological investigation in the early stages is well-nigh an impossibility, we are only familiar with the ultimate results produced. On removing the roof of the middle ear affected with the disease, the first thing that strikes one is the wideness, whiteness, and dryness of the whole cavity, the contained structures being clearly defined. Fine membranous septa in various parts can be seen, and are apparently the atrophied remains of the folds of the lining membrane. A well-marked membrane is sometimes seen running up from the tendon of the tensor tympani to the roof.

On microscopical examination the layers are atrophied, and the distinctive characteristics cannot be made out. The base of the stapes is fixed to the oval window either by calcification

or ossification of the ligamentous ring, or by deposit of new-formed osseous substance upon the inner surface of the footplate, and a complete bony union of the wall of the oval window may exist (Politzer).

The condition is thought by some to be due to a trophic lesion, as, especially in the later stages, little or no injection of the malloal vessels takes place on efficient inflation, but this may be due to the fact that the vessels share in the atrophic process, or are constricted. The fact that ganglion cells are found in the lining membrane suggests that some change in them may possibly interfere with nutrition. Trophic causes, however, will not account for bony ankylosis of the base of the stapes, a condition which points to some irritative periosteal cause. Thoma, in his work on pathology, describes an atrophic catarrh in which the mucous membrane becomes thinner and atrophied, and it must be allowed that such a process will most readily account for the condition found.

With regard to the secondary affection of the labyrinth, impairment of function may result simply from disuse, but in some cases, at all events, further changes must exist. It may be that the atrophic process is continued to the cavity of the labyrinth with resulting decrease of secretion of the intra-labyrinthine fluids, the perilymph in particular.

Some cases with symptoms closely resembling those seen in this group have been found by Toynbee, Politzer, Bezold, and others to be due to a primary affection of the labyrinthine bony capsule, producing ankylosis of the base of the stapes, without any pathological lesion of the lining membrane.

**Symptoms and Signs.**—The onset of the deafness is so insidious that, as a rule, the patient does not come for treatment until the disease is well advanced. In some a slight ringing tinnitus was present for some time before the deafness was noticed, a gradual decrease of hearing in one ear, usually the left, being unnoticed or disregarded until the other ear became seriously affected, both ears then gradually becoming worse. In other cases the patient's friends are the first to notice the diminution in function. The tinnitus is often not distressing, the patient getting absolutely used to it, in others it is one of the most prominent features. When the disease has advanced considerably in both ears paracusis is a marked symptom. The hearing is usually worse during a cold. Occasionally the disease appears to stop short, or to progress very slowly, when the later stages are reached, and complete stone deafness is never observed. On inflation a very slight improvement in hearing is produced, but soon disappears, and, as before mentioned, little or no resulting injection of the malloal vessels can be seen. In some the inflation may not be felt in the ear, although the diagnostic tube clearly indicates

that it has been successful. Attacks of true auditory vertigo are not experienced. The Eustachian tube shows no signs of obstruction, but, on the contrary, seems unusually patent, the air entering very clearly and dryly. On examination the meatus is usually clear of cerumen—in fact, patients sometimes complain that their ears seem dry. On looking at the membrane the absence of gross changes is very marked, it often looks unusually bright, clear, and thin, with little or no signs of depression. The membrane and malleus move freely with Siegle's speculum. The nose and naso-pharynx in the majority of cases appear perfectly normal, sometimes the nose may appear dry, the patient stating that a handkerchief is not often necessary, and the lining membrane of the naso-pharynx may appear thin, the lips of the Eustachian tube standing out boldly, but it is never dry and glazed. There appears to be no connection, as one would expect, between so-called atrophic rhinitis and this disease. Paleness of the soft palate with a blush on each side is usually present (Urban Pritchard).

**Prognosis.**—This is always extremely bad, no treatment has any power, apparently, to check its progress, the deafness goes from bad to worse, but may stop short at some point, absolute deafness never resulting, the patient being always able to hear something.

**Diagnosis.**—The age and sex of the patient, the insidious onset, the appearance of the membrane, the absence of Eustachian obstruction, freedom and dryness of air-entry on inflation, the slight improvement produced thereby, and absence of any cause in the nose or naso-pharynx, separate this from other middle ear diseases. The tuning-fork, etc., will distinguish it from internal, and in those cases in which internal ear results are produced by the tuning-fork the presence of paracusis will give the clue. In cases of primary disease of the labyrinthine capsule the pink lining membrane can be seen through the membrana tympani.

**Treatment.**—As before stated, we have as yet no treatment which has any power to check the disease when once it has started.

The general health of the patient must be put in the best possible condition. The local treatment is merely palliative, and even this must be used with extreme caution or the patient will be made distinctly worse. Occasional catheterisation with injection of paroline, or inflation with the bag, especially if a few drops of chloroform be previously introduced, produces a slight amount of improvement, and is a comfort to the patient. The chloride of ammonium inhaler produces little or no benefit, except that a tendency to colds is held in check.

Massage with Siegle's speculum in the ordinary way must be very cautiously and gently applied, as in the great majority of cases it is distinctly detrimental. Too prolonged

application of inflation or massage will produce undue flaccidity of the membrane, and so add to the trouble.

At the Sixth International Congress of Otolaryngology in 1899, Mink stated that he had produced good effects by using Siegle's speculum in a modified way. The membrane and the malleus are first compressed by air, stopping short of pain, before massage is applied, this method, which aims at moving the base of the stapes, has not received a fair trial.

With regard to operative intra-tympanic measures the general experience is that they are contradicted, but perhaps it is only fair to say that those on the stapes and inner middle ear wall are still on their trial. With regard to artificial aids in the extreme stage, lip-reading lessons are of great value, and may entirely alter the patient's outlook on life. Mechanical aids are mainly useful for individual conversation, when obtaining one, all varieties should be tried, as a rule, the ordinary speaking-tube will be of the greatest service.

**C CHANGES PRODUCED BY VARIATIONS IN PRESSURE — Negative Pressure in the Tympanum**—Concerning this little is known, but the adoption of a separate class for it is warranted by the deafness which occurs in those who work under increased atmospheric pressure, such as deep-sea divers, and in those cases in which deafness results as a result of chronic mechanical obstruction of the tube, such as cicatricial contraction, pressure of tumours, etc.

It may be stated, however, that it is difficult to exclude the previous forms of disease in these cases, but, on the other hand, some of the changes described as having resulted from a chronic catarrh may be due to a long-continued negative pressure.

**Causation**—These are of two varieties—

(a) Long-continued or often-repeated increase of atmospheric pressure on the membrane.

(b) Non-aeration of the middle ear, owing to—

(1) Mechanical occlusion of the tube by cicatricial contraction, pressure of tumours, etc.

(2) Nasal obstruction due to any cause, especially when affecting the inferior meatus, as this place is practically a continuation of the mouth of the Eustachian tube.

(3) Paresis of the Eustachian muscles, as occurs sometimes after diphtheria, preventing by their inaction proper aeration.

**Pathology**—Of this we have nothing but conjecture to go upon, but it can readily be conceived that if air is prevented from entering the middle ear by the atmospheric pressure from without, or by obstruction from within, a long-continued or often-repeated negative pressure in the tympanum will produce a chronic dilatation of the vessels of the lining membrane, with re-

sulting hypertrophy of the tissues and fixation of the ossicular chain.

**Symptoms and Signs**—Those of the hypertrophic class, plus the obvious cause which exists apart from catarrhal conditions.

**Prognosis**—This will depend, firstly, whether the cause can be removed, and, secondly, if removal is possible, on the results obtained by subsequent aeration of the tympanum.

**Diagnosis**—As far as can be judged at present this depends on middle ear symptoms and signs combined with an obvious cause of non-aeration apart from catarrh.

**Treatment**—In those working under increased pressure care must be taken that there is no hindrance to the entry of air through the tube. In those cases in which non-aeration is due to obstruction in the nose or naso-pharynx, removal of the cause, if possible, is indicated with subsequent aeration of the tympanum. With regard to the removal of septal spurs or hypertrophied turbinates, a good rule to observe is that they should not be interfered with unless marked blocking of the inferior meatus is present, or if the passage of the Eustachian catheter is interfered with. When once the obstruction is removed and subsequent aeration fails to produce improvement, the question of intra-tympanic operations directed to removing the rigid ossicular chain may be considered.

**D CHANGES PRODUCED BY DEFICIENT BLOOD-SUPPLY**—Of this form little definite is known, but cases are met with in the later periods of life when the clinical features—local and general—suggest that the impairment of hearing may be primarily dependent on interference with the sound-conducting apparatus due to defective nutrition of the soft structures of the middle ear. The subjective symptoms are a gradual deterioration of hearing power without tinnitus, one ear being usually more affected than the other, and varying with the general health and condition of the patient. On objective examination the membrane may be normal, but sometimes looks thinner and clearer than usual. On inflation the Eustachian tube is patent, but very slight if any improvement results, the injection of the malleal vessels after inflation being also slight. With the Siegle speculum the membrane and malleus often move well, but as a rule with no good effect. When tested with the tuning-fork it will be found that the internal ear is also impaired, in some the internal ear impairment seems to predominate. Paracusis and true auditory vertigo do not occur. (In some cases Gardiner Browne's test gives a normal result, owing to the equal impairment of both middle and internal ears, this sign, first pointed out by Urban Pritchard, is of great value.) With regard to the diagnosis of this condition, it must be admitted that it is usually difficult, even in the presence of marked evidence of general arterial disease, to clearly separate these



cases from those of senile nerve deafness (*vide* vol i. "Auditory Nerve"). The treatment in these cases is general, no local treatment is of any service.

**TESTS FOR DIAGNOSING MIDDLE EAR FROM INTERNAL EAR DISEASE**—These are chiefly of two varieties —

(a) By comparing the air and bone conduction in the patient, or with the normal, by means of a medium C tuning-fork

These tests vary in detail, but are based on the broad fact that in any external or middle ear disease bone conduction is greater than air in the patient, and is increased when compared with the normal, the converse holding in internal ear affections. The tests known as "Weber's" and "Rinne's" are those usually employed

"Gardiner Browne's" and "Schwabach's" may be of service as supplementary tests. These are described on page 459.

(b) By testing the range of tone hearing by means of tuning-forks, Galton's whistle, König's rods, and musical instruments

Broadly speaking, in middle ear disease high tones are heard better than low, and this may be carried to such an extent that the patient can hear a watch tick, and yet cannot hear thunder. In comparing watch and voice hearing, the former is often heard, comparatively speaking, better than the latter. In internal ear disease high tones are usually lost. Tuning-forks, Galton's whistle, and occasionally musical instruments, are the means used to determine these points (see p 459)



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